U.S. Department of Labor

Office of Administrative Law Judges
36 E. 7th St., Suite 2525
Cincinnati, Ohio 45202

(513) 684-3252
(513) 684-6108 (FAX)

Issue Date: 20 April 2004

Case No. 2003-BLA-25
2003-BLA-26

In the Matter of:
MARY KOURIANAKIS, Widow of
EMMANUEL KOURIANAKIS,
Claimant,

v.

PRICE RIVER COAL COMPANY, INC.,
Employer,
and
AMERICAN ELECTRIC POWER CORP.,
Carrier,

and
DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

APPEARANCES:
Jonathan Wilderman, Esq.
On behalf of Claimant

William Evans, Esq.
On behalf of Employer/Carrier

Gregory Tronson, Esq.
On behalf of Party-in-Interest

BEFORE: THOMAS F. PHALEN, JR.
Administrative Law Judge

DECISION AND ORDER – AWARDING LIVING MINER BENEFITS AND SURVIVOR BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of

the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.[1]

On September 24, 2002, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a hearing. (DX 117).[2] A formal hearing on this matter was conducted on July 17, 2003, in Price, Utah by the undersigned Administrative Law Judge. All parties were afforded the opportunity to call and to examine and cross examine witnesses, and to present evidence, as provided in the Act and the above referenced regulations.

ISSUES

The issues in the living miner claim are:

1. Whether the Miner had pneumoconiosis as defined by the Act;

2. Whether the Miner's pneumoconiosis arose out of coal mine employment;

3. Whether the Miner's disability was due to pneumoconiosis;

4. Whether the evidence establishes a material change in conditions under § 725.309(d); and

5. Whether the evidence establishes a change in conditions and/or that a mistake was made in the determination of any fact in the prior denial under § 725.310.

(DX 118). At the hearing, the parties stipulated that Miner suffered from a totally disabling respiratory or pulmonary impairment, and that Miner engaged in over thirty-years of coal mine employment. (Tr. 18, 19).

The issues in the survivor claim are:

1. Whether the Miner has pneumoconiosis as defined by the Act;

2. Whether the Miner's pneumoconiosis arose out of coal mine employment;

[1] The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

[2] In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr" refers to the official transcript of this proceeding.

3. Whether the Miner's death was due to pneumoconiosis;

(DX 119).

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

Emmanuel Kourianakis ("Miner") was born on October 24, 1922. (DX). On June 12, 1949, he married Mary (Fronimos) Kourianakis. (DX 102; Tr. 24). They lived together as husband and wife until Miner died at the age of 76 on May 13, 1998. (Tr. 24). She has not remarried. (Tr. 29). I find that Mary Kourianakis was a dependent of Miner during his lifetime for purposes of augmentation. I also find that she is an eligible surviving spouse.

Procedural History

Miner filed an initial claim for benefits under the Act on April 2, 1979. (DX 1). This claim was denied by the District Director, Office of Workers' Compensation Programs ("OWCP") on August 11, 1981. Miner took no further action on this claim. He filed his first duplicate claim on June 6, 1983. (DX 2). Miner requested a formal hearing after the duplicate claim was denied by the OWCP. After conducting a formal hearing, Administrative Law Judge Alexander Karst issued a decision and order – denial of benefits on August 4, 1986, finding that Miner failed to establish the presence of pneumoconiosis or total disability due to pneumoconiosis. (DX 48). Claimant filed an appeal with the Benefits Review Board ("Board"), but he submitted additional evidence while the appeal was pending before the Board. Thus, the Board remanded the claim to the OWCP to evaluate the new evidence as a request for modification. (DX 56). The OWCP denied Miner's request for modification on January 11, 1991. (DX 59). Claimant requested a formal hearing, and the modification request was transferred to the Office of the Administrative Law Judges. (DX 63). However, at the request of Employer, the modification request was remanded to the OWCP. (DX 64). On January 17, 1992, the OWCP issued a proposed decision and order of material change in conditions and award of benefits. (DX 68). Employer requested a formal hearing. (DX 76). After holding a formal hearing on July 12, 1995, Administrative Law Judge Daniel Roketenetz issued a decision and order – denial of benefits. Administrative Law Judge Roketenetz noted that the parties stipulated that Miner engaged in 30 years of coal mine employment, and he found Price River Coal Company to be the proper responsible operator. He then found that Miner failed to establish a material change in conditions because he didn't establish that he suffered from pneumoconiosis and that, accordingly, he could not be totally disabled due to pneumoconiosis. (DX 80). Miner filed a notice of appeal with the Board, but the Board dismissed Miner's appeal on June 4, 1996 after it determined that Miner abandoned his appeal by failing to file a petition for review and brief. (DX 84).

Miner submitted a chest x-ray interpretation to the OWCP on November 29, 1996, consisting of a radiology report and medical opinions from Dr. Guicheteau. (DX 86). Miner then submitted the report of Dr. Hill. (DX 86). The OWCP considered Miner's submission of medical evidence as a modification request. In a letter dated January 14, 1997, the OWCP notified Miner that the medical evidence he submitted was deficient because the November 19, 1996 x-ray was not interpreted by a B-reader. The OWCP requested that Miner submit the film from the November 19, 1996 x-ray. Miner was granted 45 days to complete the request of the OWCP. (DX 87). On February 6, 1997, Miner submitted a re-reading of the November 19, 1996 film by Dr. Preger, but Dr. Preger noted that his reading of the November 19, 1996 film was from a copy of the film. (DX 88). The OWCP issued a proposed decision and order denying request for modification on March 3, 1997. (DX 92). The OWCP received additional medical evidence on March 31, 1997 and April 7, 1997. (DX 98). The OWCP filed a request for modification on April 7, 1997. (DX 98). Miner died on May 13, 1998.

Mary Kourianakis ("Claimant") filed a claim for survivor's benefits on July 28, 1998. (DX 99). On December 29, 1999, the OWCP issued an order to show cause why modification should not be granted. (DX 96). The OWCP issued a proposed decision and order approving request for modification and award of benefits on Miner's living miner claim on May 3, 2000. (DX 98). Employer requested a formal hearing before the Office of the Administrative Law Judges. Employer filed a motion to dismiss on July 3, 2003.[3] Claimant submitted a letter stating her objection to Employer's motion to dismiss and the OWCP filed a statement in opposition to Employer's motion to dismiss. A formal hearing was conducted on July 17, 2003. The OWCP presented testimony from Joseph Contardo, who was a senior claims examiner employed by the OWCP in Denver, Colorado office where Miner's claim was processed. (Tr. 106). The undersigned issued an order denying Employer's motion to dismiss on August 28, 2003. Employer requested that the undersigned reconsider the order denying the motion to dismiss. On October 6, 2003, the undersigned issued an order denying Employer's motion for reconsideration. Employer filed a notice of appeal on November 5, 2003 with the Board, which the Board acknowledged on November 24, 2003. The Board issued an order on January 14, 2004, dismissing Employer's appeal because it was interlocutory in nature and did not meet the three-prong test for the limited circumstances when the Board would hear an interlocutory appeal.

Length of Coal Mine Employment

The parties stipulated that Mr. Kourianakis was a coal miner within the meaning of § 402(d) of the Act and § 725.202 of the regulations, and that Mr. Kourianakis engaged in more than 30 years of coal mine employment. The stipulations are supported by the evidence of record. Therefore, I find that Miner engaged in coal mine employment for at least 30 years.

[3] The undersigned incorporates herein the findings set forth in the orders dated August 28, 2003 and October 6, 2003. In those two orders, the Employer's motion to dismiss is erroneously identified as having been filed on July 7, 2000. It was actually filed on July 3, 2003.

Responsible Operator

Liability under the Act is assessed against the most recent operator which meets the requirements of § 725.493(a)(1). The District Director identified Price River Coal Company, Inc. as the putative responsible operator. Price River Coal Company, Inc. stipulated that it is the employer with whom Mr. Kourianakis spent his last cumulative one year period of coal mine employment and that it is properly designated as the responsible operator in this case. § 725.493(a)(1). The stipulation is supported by the evidence of record that establishes that Miner worked for Price River Coal Company for 17 years, ending in 1982. Miner did not engage in coal mine employment after leaving Price River Coal Company. I find that Price River Coal Company is the proper responsible operator.

MEDICAL EVIDENCE

I incorporate by reference, as if fully rewritten herein, the chest x-ray interpretations, pulmonary function tests, arterial blood gas studies, and narrative medical evidence contained in the December 5, 1995 decision and order – denial of benefits issued by Administrative Law Judge Daniel Roketenetz and the August 4, 1983 decision and order – denial of benefits issued by Administrative Law Judge Alexander Karst, to the extent the evidence is not inconsistent with the evidence summarized herein.

X-RAY REPORTS

Exhibit	Date of X-ray	Date of Reading	Physician/Qualifications	Interpretation
DX 94	11/19/95	3/28/97	Gotthoffer, BCR	1/1
DX 95	11/19/95	4/9/97	Preger, BCR[4], B-reader[5]	1/0; film quality 2

Narrative Medical Evidence

Rodney Badger, M.D. admitted Miner to Utah Valley Regional Medical Center on December 19, 1995, after Miner experienced a recent episode of chest pain and shortness of breath with coughing. (DX 106). An adenosine stress test was positive for ischemia. Dr. Badger noted that Miner had a history of cigarette smoking hypercholesterolemia and hypertension with chronic obstructive pulmonary

[4] A physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. *See* 20 C.F.R. § 727.206(b)(2)(III). The qualifications of physicians are a matter of public record at the National Institute of Occupational Safety and Health reviewing facility at Morgantown, West Virginia.

[5] A "B" reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. This is a matter of public record at HHS National Institute for Occupational Safety and Health reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.5l) Consequently, greater weight is given to a diagnosis by a "B" Reader. *See Blackburn v. Director, OWCP*, 2 B.L.R. 1-153 (1979).

disease ("COPD") and silicosis. His plan was to submit Miner to a left and right heart catheterization with coronary angiography. Dr. Badger's impression after the catheterization was that Miner suffered from severe pulmonary hypertension, possible mitral stenosis, and mild elevation of left ventricular, end diastolic, and pulmonary capillary wedge pressure. Miner was discharged by Dr. Hill on December 20, 1995.

Kurt King, M.D. examined Miner on December 19, 1995 after being asked by Miner to evaluate him for the presence of black lung. (DX 106). He noted that Miner had been admitted to Utah Valley Regional Medical Center for a heart catheterization that was performed by Dr. Badger and showed a normal left ventricle, a question of mitral stenosis, and severe pulmonary artery hypertension. Dr. King considered that Miner had a 38 year history of mostly above ground coal mine employment, as well as a 40 pack-year smoking history from 1942 through 1982. Dr. King noted that Miner experiences shortness of breath on approximately 30 yards exertion and has less than one flight of stair dyspnea on exertion. On physical examination, Dr. King found Miner's chest to be barrel in nature, with prolonged expiration, decreased breath sounds, but no wheezes. He did not have any laboratory data relevant to evaluating Miner for black lung at the time of his examination. Dr. King's impression was severe pulmonary disease, probably mixed chronic obstructive pulmonary disease with some elements of black lung (no proof of that at this time), severe pulmonary hypertension, and social problem due to black lung compensation issues as discussed in his report.

Dr. Douglas Wing interpreted a portable chest x-ray, obtained on December 20, 1995, as revealing cardiomegaly, prominent lung markings in both bases (which may be old scarring or perhaps interstitial edema, but he favored scarring), fractures of the posterior 6th, 7th, and probably 8th ribs, as well as old granulomatous disease. (DX 106). Dr. Wing interpreted a portable chest x-ray obtained on December 23, 1995 as showing posterior left rib fractures and no pneumothorax. (DX 106).

John Guicheteau, M.D. examined Miner on November 19, 1996. (DX 86). He considered a 42 pack-year smoking history and a coal mine employment history of 33 years. Dr. Guicheteau noted that Miner was on continuous oxygen and had two episodes of pneumonia in 1994 and 1995. Miner reported dyspnea after 50 feet and a cough producing up to one-half cup of sputum per day. On physical examination, Dr. Guicheteau found increased AP diameter in Miner's chest, some decreased diaphragmatic excursion, pan inspiratory crackles in both bases, late inspiratory crackles in the right mid lung field, and mid-to-late wheezing in both lung fields. Dr. Guicheteau rendered the following appraisal: (1) underlying respiratory disease – COPD, reactive airway disease, prior tobacco history, and mining exposure (will review chest x-ray, PFTs, ABGs, to determine potential etiology of disability); (2) atherosclerosis; (3) hearing aids; (4) prior cataract surgery and corneal implant; (5) prior pneumonia; (6) history of knee infection; (7) prior appendectomy; (8) penicillin allergy; (9) war injury '44 for right arm shell wound and absent right bicep reflex; (10) bilateral toe amputation '52/53; and (11) evidence of cor pulmonale based on hepatojugular reflux, will review EKG and chest x-ray. Dr. Guicheteau stated that he will review all of the data and then dictate a letter outlining the etiology of Miner's underlying lung disease. Attached to his report were the results of an ABG conducted on November 19, 1996 that would establish a presumption of total disability, a chest x-ray interpreted by Dr. Gotthoffer as 1/1 for pneumoconiosis, and an EKG printout.

Jeffrey McCellan, M.D. interpreted a CT scan of Miner's thorax on December 2, 1996. (DX 106). His impression was mild interstitial thickening of the lung bases that is nonspecific with very

slight increase in the density of the lung parenchymal and a vague-type pattern which can be seen with alveolitis. He also found the CT scan to be negative for mediastinal or hilar adenopathy. Dr. Rodney Petersen added an addendum to the CT scan interpretation of Dr. McCellan on December 3, 1996. His impression was: (1) there is some calcified hilar nodes bilaterally as well as what appear to be some peripheral granulomata. The findings are consistent with old granulomatous disease with hilar calcifications also being related to a pneumoconiosis such as silicosis; (2) there are interstitial changes with a mosaic pattern predominantly in the mid and lower lung fields, some subtle irregular opacities, again most common inferiorly and posteriorly. These are nonspecific but are not inconsistent with a pneumoconiosis such as anthracosis; and (3) No significant pleural changes are noted. No definite acute abnormalities could be seen.

Tracy Hill, M.D. issued a narrative report on December 4, 1996. (DX 86). Dr. Hill stated that he had been asked by Miner to render an opinion concerning the possibility that Miner suffers from black lung. Dr. Hill considered a 38 year history of coal mine employment that was mostly above ground. He also considered a 40 pack-year smoking history from 1942 through 1982. Dr. Hill noted that Miner suffers from significant pulmonary limitation, including oxygen dependency and marked shortness of breath, which was documented by the PFT he conducted on December 20, 1995. He opined that the following evidence supports a finding that Miner's coal dust exposure contributed to some degree to his pulmonary limitation: (1) appropriate exposure to coal dust; (2) CT scan dated December 2, 1996 that showed some evidence of small opacities and hilar calcification consistent with coal-dust injury to the lung; and (3) evidence in published studies that suggested that the effects of mine-dust exposure is approximately one-third as severe as the effects of cigarette smoking. Dr. Hill stated that Miner has a severe reduction in his FEV1 value. He found it reasonable to attribute some of Miner's FEV1 limitation to coal-dust exposure. Dr. Hill concluded that Miner's severe pulmonary limitation has clearly been significantly contributed to by Miner's smoking history. However, he stated that there is reason to believe that there is some contribution from coal dust exposure. He was unable to provide an opinion on the precise percentage that coal dust exposure contributed.

John Guicheteau, M.D., who is board-certified in internal medicine, issued a narrative report on December 21, 1996. (DX 86). He examined Miner on November 19, 1996. He opined that Miner had simple pneumoconiosis based on his chest x-ray with a history of 33 years of coal mine employment and a 40 pack year history of cigarette smoking. Dr. Guicheteau opined that Miner is permanently and totally disabled from a pulmonary standpoint based on the resting arterial blood gas studies, pulmonary function tests, chest x-ray, physical examination, and history. He noted that an adenosine stress test failed to identify underlying ischemia. He opined that the etiology of Miner's disability was pneumoconiosis and cigarette smoking. Dr. Guicheteau documented references to pneumoconiosis by Drs. King, Taylor, and Gotthoffer. He opined that Miner's smoking history does not negate the aspect of 33 years of coal dust exposure contributing to Miner's disability. Dr. Guicheteau concluded that Miner's pneumoconiosis was a presumptive contributing cause to his pulmonary disability as was his coal dust exposure. He stated that a lung biopsy was the manner for determining the presence of pneumoconiosis, but he advised that Miner should not undergo a lung biopsy due to the intrinsic risks involved. Dr. Guicheteau opined that it is difficult to prove the absence of pneumoconiosis as a contributing factor to Miner's disability without biopsy evidence.

Miner's certificate of death, listing his date of death as May 13, 1998, was signed on May 13, 1998 by Dr. Sterling Potter. (DX 103). Dr. Potter identified the immediate cause of death as cardiac

arrest, with underlying causes of cardiomegaly, chronic cor pulmonale, and chronic obstructive pulmonary disease ("COPD"). The autopsy findings were not available to Dr. Potter when he signed the death certificate.

Leo Hardy, M.D., who is board-certified in clinical and anatomical pathology, conducted an autopsy limited to Miner's chest on May 13, 1998. (DX 104). Dr. Hardy considered a clinical history of congestive heart failure, hypertension, osteopenia/osteoarthritis, history of smoking and occupational exposure to coal dust. Dr. Hardy also noted detailed findings of Miner's final hospitalization at Castleview Hospital from April 21, 1998. Dr. Hardy provided a gross description of Miner's overall appearance. He documented the surgical technique he used to exam Miner's chest. Dr. Hardy provided macroscopic and microscopic findings and observations of Miner's heart and lungs. In Miner's lungs, Dr. Hardy identified multiple anthracotic lymph nodes. He detected bullous emphysematous changes that progressively decrease inferiorly characteristic of centri-lobular emphysema. He also found emphysematous anthracotic changes that were moderate-to-severe throughout that decreased in the inferior lobes. Dr. Hardy rendered a final anatomical general finding of atherosclerosis, generalized, moderate. His final findings regarding Miner's heart were: (1) cardiomegaly; (2) right atrial dilation and right ventricular hypertrophy, severe (cor pulmonale); (3) left ventricular hypertrophy, moderate-severe; (4) ischemic heart disease, moderate; (5) mitral valve calcifications, mild; (6) coronary arteriosclerosis, mild-moderate; and (7) history of hypertension. Dr. Hardy's final anatomic lung findings were: (1) anthracosilicosis, and micronodules (5 mm), moderate (simple coal workers' pneumoconiosis ("CWP")); (2) centrilobular emphysema, severe; (3) pleural adhesions, mild; (4) pulmonary hypertension, severe; (5) diffuse alveolar damage, organizing pneumonia, and bronchiolitis obliterans, focal (right middle lobe, right inferior lobe, and left inferior lobe); and (6) pleural effusions, serous, bilateral. He also listed other diagnoses of actinic keratoses, missing second digits, osteoarthritis, venostasis. Dr. Hardy concluded that dysrrhythmia secondary to ischemic heart disease was possibly the underlying cause of death. Dr. Hardy also prepared a final note, reiterating his findings. He noted that the limited autopsy showed moderate anthracosilicosis, micronodules, and severe centrilobular emphysema. Under high power microscopy, Dr. Hardy determined that the anthracosilicotic pigment showed variable morphology, some of which had the appearance of coal dust (translucent, golden, and larger) and some of which was consistent with combustion pigments (black, opaque, and smaller). Dr. Hardy stated that he found anthracotic pigment macules and micronodules up to 5 cm. In the areas of pigment deposition, polarized light microscopy showed characteristic delicate silica particles. Dr. Hardy stated that features of progressive massive fibrosis were not found. He was unable to determine the immediate underlying cause of death. Given the degree of Miner's ischemic heart disease, Dr. Hardy found that the possibility of dysrhythmia was likely. He could not exclude the possibility of an intracranial process or other etiology, such as a gastric/colonic bleed. Dr. Hardy again noted that the lungs showed severe emphysematous changes and findings consistent with simple CWP.

Joshua Perper, M.D., who is board-certified in clinical, anatomical, and forensic pathology, issued a narrative opinion on August 30, 1999. (DX 105). He reviewed and summarized Miner's employment records, death certificate, autopsy report and slides, hospital records, and chest x-ray reports. Dr. Perper considered a coal mine employment history of 38 years in various capacities, such as boney picker, trackman, tipple operator, and general laborer. He also considered a 40 pack year smoking history from 1942 through 1982. Dr. Perper rendered the following microscopic diagnoses from the autopsy slides: (1) CWP, slight, with macules, micronodules, silica crystals, focal emphysema, and isolated silicotic peri-bronchial lymph node; (2) centrilobular emphysema and interstitial fibrosis,

moderate to marked; (3) foci of organizing and organized pneumonia; (4) foci of intra-pulmonary bony metaplasia, few; (5) sclerosis of small intra-pulmonary blood vessels, severe, indicative of pulmonary hypertension; (7) slight to moderate arteriosclerosis of aorta and coronary arteries; and (8) focal myocardial fibrosis, minimal. Dr. Perper opined that Miner had evidence of slight-to-moderate simple CWP based on: (1) more than sufficient exposure history; (2) respiratory symptomatology with shortness of breath on exertion, cough, and expectoration of sputum; (3) severe objective respiratory dysfunction with disturbed obstructive ventilatory tests, hypoxemia, marked decrease in pulmonary diffusion and oxygen saturation requiring continuous administration of oxygen and bronchodilators, and evidence of severe pulmonary hypertension measured clinically with radiological evidence of pulmonary hypertension. Evidence at autopsy of severe cor pulmonale and arteriosclerosis of intra-pulmonary blood vessels indicative and consistent with the clinical findings of severe pulmonary hypertension; (4) clinical findings of obstructive pulmonary disease with barrel shaped chest, decreased breathing sounds, and distal heart sounds. Even though the radiologists may have missed a diagnosis of CWP, it was not surprising because scientific literature establishes that mild CWP cases are missed and even up to one-third of complicated cases are missed; and (5) the autopsy showed the presence of mild CWP with primarily macular lesions and small numbers of micronodules, as well as the presence of interstitial fibrosis with mixed anthracotic pigmentation and multiple silica crystals in the alveolar septa and severe associated centrilobular emphysema. Dr. Perper noted that emphysema is known to be associated with exposure to coal mine dust containing silica as well as exposure to cigarette smoke. He added that the combination of Miner's heavy smoking does not and cannot exclude the equally substantial contribution of significant exposure to coal mine dust containing silica. Dr. Perper cited to scientific literature to support his conclusion that Miner's centrilobular emphysema was causally associated with his coal mine dust inhalation. He opined that Miner's CWP was a substantial contributory cause of Miner's death based on the significant presence of CWP and associated centrilobular emphysema. Moreover, the marked focal fibrosis associated with anthracotic pigmentation (and the silica crystals) was clearly due exclusively to the pneumoconiotic process as the fibrous anthracotic septa contained multiple silica crystals. Dr. Perper stated that "the most likely mechanisms of death by which pneumoconiosis contributed to death was both hypoxemia that triggered or aggravated an arrhythmia caused by Miner's ischemic heart disease. He emphasized that Miner's coronary arteriosclerotic heart disease was only slight-to-moderate, and overshadowed by Miner's pulmonary disease. Dr. Perper then reiterated his findings in three conclusions. First, he stated that the primary cause of death was a result of combined heart disease with cardiomegaly and severe COPD with simple CWP. Second, Dr. Perper found that Miner had evidence of simple CWP with associated severe centrilobular emphysema and pulmonary fibrosis, severe pulmonary hypertension, and cor pulmonale. He added that Miner's severe centrilobular emphysema was, in part, a result of Miner's heavy smoking exposure. Third, Dr. Perper concluded that Miner developed CWP as a result of long occupational exposure to coal mine dust. Lastly, Dr. Perper found that Miner's CWP was a substantial contributory cause of his death. Dr. Perper attached an appendix identifying scientific literature that establishes the existence of a causal relationship between coal mine dust inhalation and centrilobular emphysema.

Richard Naeye, M.D., who is board-certified in clinical and anatomical pathology, issued a narrative medical report on November 19, 1999. (DX 13). He reviewed the autopsy report, the death certificate, 21 autopsy slides, as well as hospital records and physician reports. Dr. Naeye considered a coal mine employment history of 38 years ending in 1982. He documented a smoking history of one pack per day for 40 years, also ending in 1982. Dr. Naeye recorded a clinical history of hypertension, congestive heart failure, chronic bronchitis, emphysema, gastric ulcers, and senile dementia. Dr. Naeye

noted that death was attributed to a combination of cardiac failure, chronic cor pulmonale, and COPD. Dr. Naeye rendered microscopic findings from the ten slides containing lung tissue. He found a small-to-moderate amount of black pigment in the lung tissue, which was almost entirely in deposits .1-.3 mm in diameter. The areas were adjacent to small airways, small arteries, and in the subpleural spaces. Dr. Naeye detected a small amount of fibrous tissue associated with the black pigment at some sites, but no admixed tiny birefringent crystals that might be free silica. He also found three sites where the anthracotic deposits reached .4, .10, and .10 mm in diameter. He found rims of focal emphysema around some of the deposits. Dr. Naeye also detected severe centrilobular emphysema. He noted that Miner's heart was enlarged at autopsy, with the left ventricle wall measuring 22 mm in thickness and the right ventricle wall measuring 12 mm in thickness. He opined that the 12 mm measurement is not the result of CWP causing cor pulmonale because the CWP is far too mild to cause this effect. Instead, he attributed it to Miner's severe centrilobular emphysema, chronic hypertension, and resulting left heart failure, which increased pulmonary venous blood pressure. Dr. Naeye opined that Miner's simple CWP was far too mild to have caused structural abnormalities in Miner's lungs that would have resulted in chronic cor pulmonale. Dr. Naeye concluded that Miner had postmortem lung tissue findings of mild, simple CWP, severe centrilobular emphysema, and chronic bronchitis. He stated that the absence of radiological evidence of CWP was indicative that Miner's CWP was mild. The mild nature of Miner's CWP led Dr. Naeye to conclude that CWP did not produce any disability during Miner's life, nor did it hasten Miner's death. Dr. Naeye noted that his CWP was not complicated because the 1 cm lesions he found were formed years before Miner died and may not be solely occupational in origin.

Dr. Naeye then addressed the issue of whether Miner's severe centrilobular emphysema and resulting COPD were the result of Miner's occupational exposure or wholly the consequence of Miner's cigarette smoking. He stated that centrilobular emphysema has, without a doubt, had a partial occupational origin in anthracite and medium rank bituminous coal miners in eastern and central Pennsylvania. However, he added that a critical review of medical literature indicates that mine dust has no role or only an insignificant role in the genesis of centrilobular emphysema. He referenced medical literature and studies regarding the relationship of coal dust exposure and cigarette smoking to centrilobular emphysema. Dr. Naeye opined that Dr. Perper's review of the medical literature was based in large part on selective European studies, and does not take into consideration the complicated interaction of cigarette smoking on the outcome of epidemiologic studies. He reiterated his statement that anthracite and medium rank bituminous coal mine dust is toxic in the United States, and he added that, due to the variability of the toxicity of coal mine dust, some British soft-coal miners have centrilobular emphysema as the result of their occupational exposures. Dr. Naeye mentioned that he only knew of one coal seam in the US soft coal regions of western Pennsylvania, West Virginia, Virginia, Kentucky, the mid-west, Wyoming, Colorado, and Utah where the mine dust has the toxicity to cause centrilobular emphysema. He then stated that "[i]n the past years I reviewed the lungs of many miners of this coal seam and regularly recognized that their exposures to dust had made a major contribution to their emphysema and disability." Dr. Naeye added that Miner was not one of those miners. He determined that Miner was disabled in his late years by a combination of severe centrilobular emphysema, chronic bronchitis, cardiac failure, and dementia. Dr. Naeye considered Miner's CWP to be far too mild to have any contribution to Miner's disability and was also far too mild to have hastened or had any other role in Miner's death.

Kurt King, M.D., who is board-certified in internal medicine, issued a narrative opinion on June 20, 2003. (CX 3). He stated that he reviewed his office notes regarding Miner, along with pulmonary,

neurology, and cardiology consults that were obtained over the years. He spoke with Dr. Hardy and reviewed Dr. Hardy's autopsy report. Dr. King noted that he was Miner's treating physician from August 6, 1991 until October 8, 1997. He stated that he saw Miner four to six times per year. Dr. King opined that Miner's main disability was emphysema and pulmonary hypertension, mostly caused by cigarette use, with coal dust exposure being a significant contributing factor to the development of the emphysema. He felt that Miner's CWP, proven by autopsy, contributed to Miner's disability while he was alive and also contributed to his death. Dr. King added that Miner's CWP caused nodule formation (Dr. Hardy considered it to be moderate), which alters the normal lung architecture. Dr. King found that these nodules contributed to Miner's hypoxia in conjunction with the emphysema. He stated that the hypoxia and altered lung structure led to pulmonary hypertension. Dr. King determined that Miner's heart was damaged by his pulmonary hypertension, causing it to dilate and hypertrophy (findings that were identified at autopsy). He concluded that Miner's damaged heart was less able to handle an ischemic insult. Dr. King determined that the main cause of Miner's disability and death was his emphysema and pulmonary hypertension, with CWP serving as a contributory factor.

On June 23, 2003, Dr. Perper submitted a follow-up report to his August 30, 1999 report. (CX 1). He reviewed and summarized his initial report, the autopsy report and slides, the death certificate, Dr. Naeye's November 19, 1999 repot, Dr. Preger's interpretations of the x-rays dated June 26, 1995 and November 19, 1996, Dr. Gotthoffer's interpretation of the November 19, 1996 x-ray, Dr. Guicheteau's November 19, 1996 report, Dr. Hills' December 4, 1996 report, the records from Castleview Hospital, arterial blood gas studies from 1985 through 1995, and pulmonary function tests from 1983 to 1990. He stated that a repeated review of the autopsy slides did not reveal any additional findings. After summarizing Dr. Naeye's report, Dr. Perper provided comments concerning his opinion that Dr. Naeye's opinions are in error. He found that Dr. Naeye minimizes with no justification the severity of the CWP present in Miner's lungs as substantiated by the autopsy. Dr. Perper commented that he and Dr. Hardy found the CWP present to be slight-moderate. He added that Dr. Naeye himself even found two macronodules measuring 1.0 cm, which is a significant finding. Dr. Perper added that it is unclear why Dr. Naeye did not find birefringent silical particles, since both he and Dr. Hardy found them. He found Dr. Naeye's review of the clinical data to be too superficial, noting that he omitted the diagnosis of CWP by a number of clinicians based on the totality of clinical, radiological, PFT, and ABG evidence. Dr. Perper discounted Dr. Naeye's reliance on the negative radiological evidence to serve as strong support for the physiological insignificance of Miner's CWP for two reasons: (1) pathology evidence is the gold standard for diagnosis CWP; and (2) personal experience and scientific data shows that radiologists often miss a diagnosis of significant CWP, adding that a few radiologists who interpreted the presence of granulomas mistook CWP for granulomas because no granulomas were found during the autopsy. Dr. Perper opined that Dr. Naeye relied on medical literature that is virtually obsolete to state that coal dust exposure and CWP does not result in centrilobular emphysema except in Europe and virtually only in the presence of complicated pneumoconiosis. Dr. Perper found that the fact that Miner stopped smoking for years before his demise served as reinforcing evidence of the causal relationship between Miner's exposure to mixed coal dust containing silica, Miner's CWP, and Miner's COPD. Moreover, the fact that Miner's COPD and the appearance of pulmonary opacities, which worsened years after Miner stopped smoking, reinforces the predominant role of CWP in the causation of significant COPD because: (1) cessation of smoking stops any progression of smoking related centrilobular emphysema; and (2) cessation of occupational exposure does not stop the relentless process of CWP and related centrilobular emphysema because of the toxic effects of the mixed coal dust entrapped in the lungs. Dr. Perper again concluded that the medical evidence establishes the presence of

both medical and legal CWP. He stated that Miner's CWP, based on its association with Miner's hypoxemia and the causal association between coal dust and centrilobular emphysema, establish that Miner's totally disabling respiratory disability was contributed to by Miner's CWP. Dr. Perper reaffirmed his finding that Miner's death was contributed to and/or hastened by CWP, which was based on Miner's long occupational exposure to coal mine dust, Miner's respiratory symptomatology, the objective evidence of primarily obstructive lung disease and hypoxemia, and the resulting cor pulmonale with evidence of severe pulmonary hypertension, the significant CWP demonstrated at autopsy, and the associated severe centrilobular emphysema. He stated that there was competent evidence, both direct and indirect, that Miner's CWP was a significant contributory cause of death and a hastening factor based on: (1) direct replacement of normal lung tissue by pneumoconiotic lesions and associated centrilobular chronic emphysema and resulting hypoxemia, which was also demonstrated clinically; (2) the mechanism of death contributed to by the presence of CWP was through a direct pulmonary insufficiency of normally breathing lung by non-breathing pneumoconiotic tissues and associated centrilobular emphysema and resulting hypoxemia, and through hypoxemia precipitating/aggravating a cardiac arrhythmia in an individual with heart disease. He referenced a scientific article that noted that patients with COPD, especially during acute exacerbations of their disease, show a greater incidence of cardiac arrhythmia than healthy subjects of the same age.

Lawrence Repsher, M.D., who is board-certified in internal medicine and the subspecialty of pulmonary disease, issued a consultative report on June 25, 2003. (EX 5). He considered that Miner was a 76 years old at the time of his death, with a 38 year history of working in underground and surface coal mining. He also considered a cigarette smoking history of one pack per day from 1942 to 1982. Dr. Repsher summarized medical records of Miner, which included the death certificate, autopsy report, and the medical opinions of Drs. Hill, Perper, and Naeye. Dr. Repsher provided the following impression: (1) no radiographic CWP, but does have mild histologic simple CWP, probably of no clinical significance; (2) COPD, with severe centrilobular emphysema, and severe cor pulmonale, as the result of his long and heavy cigarette smoking habit; (3) hypertension, with hypertensive cardiovascular disease, with probably diastolic congestive heart failure; (4) peptic ulcer disease, with recurrent upper GI hemorrhage; (5) dementia, secondary to diagnosis #3, possibly due to diagnosis #6; (6) coronary artery disease, with possible old myocardial infarction; (7) hyperlipidemia; and (8) terminal bacterial pneumonia and ARDS. Dr. Repsher then opined, based on his findings, that Miner "did not suffer from during his life, nor was his death caused by, contributed to, or even hastened by his mild histological simple CWP that resulted from the inhalation of coal mine dust, while working for Kaiser Steel as a coal miner." Dr. Repsher then provided the following reasons to support his conclusion: (1) Miner only had very mild histological CWP that was not even radiographically visible; (2) Miner's death certificate did not even mention CWP; and (3) Drs. Perper and Naeye agreed that Miner only had slight or moderate histologic CWP. Thus, the amount of lung tissue involved would have been so small that it would not be anticipated to significantly affect Miner's overall lung function. Since there was no significant effect on lung function, then it would not be anticipated to a medical certainty that Miner's mild histologic CWP would have affected his lung function during life, nor would it be anticipated that it caused, contributed to, or hastened Miner's death. Dr. Repsher concluded that Miner's death was clearly due to many of the overwhelming deleterious effects of cigarette smoking, and in no individually discernable way by his mild histologic CWP.

Dr. Naeye submitted a supplemental narrative medical report on June 26, 2003 in response to the criticisms of his report levied by Dr. Perper. (EX 3). Dr. Naeye addressed three specific criticisms.

First, he stated that he rejects a diagnosis of complicated pneumoconiosis based on the composition of the largest lesion(s). He determined that the centers of the largest CWP lesions are not necrotic, which is inconsistent with a finding of complicated CWP lesion. More importantly, Dr. Naeye noted that the growing edge of complicated CWP lesions have chronic inflammatory cells and fibroblasts. He stated that these findings were absent in the micronodules of the lungs of Miner. Thus, he opined that the lesions which Dr. Perper suggested may be complicated CWP are actually micronodules of simple CWP. Dr. Naeye's second response to Dr. Perper's criticism was a reiteration of his finding that the very tiny birefringent crystals of toxic silica that cause fibrosis and lung damage were absent in Miner's lungs. He did find a few larger crystals, but their size identified them as non-toxic silicates. Dr. Naeye asserted that silicates do not produce fibrosis or any other tissue damage in lungs. The third criticism that Dr. Naeye addressed concerns "[t]he clinical, arterial blood gas and pulmonary function abnormalities to which Dr. Perper refers were the consequence of 40 pack years of cigarette smoking." Dr. Naeye cited a scientific study that supported this conclusion. He noted that it was important that Miner spent most of his 38 years of coal mine employment in the industry above ground where exposure to coal dust is high and exposure to mine dust is nil. He also noted that Miner always worked away from the coal face on the belt line hauling materials. Dr. Naeye noted that the study he referenced found that, for those who worked underground and away from the coal face, the effects of cigarette smoking were three times that of mine dust. He noted that Miner was exposed to very little if any mine dust, which sometimes contains toxic free silica. Rather, Miner was primarily exposed to coal dust, which is not toxic because it contains little, if any, free silica.

Dr. Repsher testified at the hearing on July 17, 2003. (Tr. 51). He stated that he was board-certified in internal medicine and the subspecialty of pulmonary disease. He identified the medical records that he reviewed prior to issuing his June 25, 2003 narrative medical opinion, which included the autopsy report and Dr. Perper's reports. Dr. Repsher testified that he disagreed that Dr. Preger's April 9, 1997 interpretation (1/0, t, t) of the chest x-ray film obtained on November 19, 1996 was positive for the presence of pneumoconiosis. Instead, he opined that Dr. Preger's findings were consistent with asbestosis and the classic changes of cigarette smoking. He stated that CWP produces rounded opacities (p, q, and r), not isolated lineal opacities (s, t, and u). Dr. Repsher testified that he agreed with Dr. Perper's finding of COPD. He also agreed that Miner suffered from simple CWP on the basis of his review of the reports of three pathologists of record. Dr. Repsher also agreed that Miner suffered from centrilobular emphysema. He testified that there is no credible published literature that shows a person can develop clinically significant centrilobular emphysema solely from exposure to coal dust. Dr. Repsher reviewed the literature Dr. Perper cited to in support of his opinion that Miner's coal dust exposure contributed to the development of his centrilobular emphysema, and he found that Dr. Perper's opinion did not accurately reflect the findings of those articles because some of the articles didn't even document that there was coal dust related centrilobular emphysema. He also opined that the majority of the studies did not control for smoking, which is the most likely cause of centrilobular emphysema. Dr. Repsher then reiterated his findings that Miner suffered from clinically insignificant lung pathology and very severe COPD that was complicated by centrilobular emphysema and cor pulmonale. He noted that Miner also suffered from coronary artery disease, which Dr. Repsher stated was probably the actual cause of death. Dr. Repsher characterized Miner's COPD as his major medical problem that forced Miner to retire and caused his disability during his lifetime. He found that Miner's COPD, centrilobular emphysema, and cor pulmonale were caused by Miner's smoking history and not by his coal dust exposure. Based on the description of the pathologist, Dr. Repsher considered that Miner's CWP involved only a very small fraction of Miner's lung tissue. Thus, it would not cause any measurable

effect on Miner's lung function. Dr. Repsher found that Miner's CWP was not clinically significant or relevant. He testified that Miner died during his sleep. Given Miner's underlying coronary artery disease, he would find that Miner either died as a result of a heart attack or a malignant arrhythmia (a fatal irregular beating of the heart). Dr. Repsher answered that Miner's pulmonary disability did not have any relation to his heart attack or heart disease. He testified that cigarette smoking and heredity are the common causes of the type of heart disease Miner had. He did agree that it is possible, but not probable, that Miner's arrhythmia may have been triggered by an event of hypoxia caused by Miner's severe COPD. Dr. Repsher testified that Miner's "histological coal workers' pneumoconiosis" was not a contributor to Miner's heart disease. He also testified that the most common cause of cor pulmonale is left ventricular heart disease, which is associated with coronary artery disease.

On cross-examination, Dr. Repsher noted that he never examined Miner, didn't review the autopsy slides, nor did he ever render an interpretation of a chest x-ray film obtained from Miner. Dr. Repsher recalled his report finding that Miner's cor pulmonale was caused by his COPD. He testified that he was aware that Dr. Hardy described Miner's simple CWP as moderate. Dr. Repsher stated that he was not aware when he rendered his narrative report that the autopsy results were not available to the physician who signed Miner's death certificate. He acknowledged that his report was based on Dr. Perper's summary of the medical records because he did not review the actual medical records. At the time he issued his narrative report, he had not reviewed Dr. Preger's interpretation of the November 19, 1996 x-ray. Counsel for Claimant offered into evidence an article published by the Department of Health and Human Services, NIOSH, entitled "A criteria for recommended standard occupational exposure to respirable coal mine dust" dated September 1995. Dr. Repsher acknowledged that the report references studies, specifically a study by Cochroft, that found coal dust contributed to centrilobular emphysema after accounting for miners and non-miners who had died of ischemic heart disease and after accounting for age and smoking history. However, Dr. Repsher testified that these articles were not credible and that they didn't actually control for cigarette smoking. He also testified that these papers did not distinguish the forms of emphysema: centrilobular emphysema versus focal emphysema, the latter of which is associated with coal dust inhalation. He found this difference to be significant because focal emphysema is not generally associated with lung function abnormality, while centrilobular emphysema is associated with catastrophic impairment of lung function.

On re-direct examination, Dr. Repsher identified a report entitled "Pathology Standards for Coal Workers' Pneumoconiosis", which he identified as containing the standards for diagnosing pneumoconiosis and complicated pneumoconiosis by pathology evidence. Dr. Repsher noted that silica, which is the most toxic of the crystals, is weakly birefringent and very small and faint when viewed under a microscope. He referenced the opinions of Drs. Naeye and Perper, noting that both found silicate crystals that can be found in anyone. He then identified Dr. Naeye's point that the one centimeter lesions in Miner's lungs did not contain anthracotic pigment, nor did they contain the silica crystals required to meet the histologic criteria for diagnosing a complicated lesion of pneumoconiosis. Dr. Repsher testified that Dr. Perper did not distinguish in his report between the weakly birefringent crystals of toxic silica and the strongly birefringent silicate crystals. He then stated that Drs. Naeye and Perper did not find the presence of weakly birefringent toxic silica crystals. Dr. Repsher testified that Miner's CWP did not cause any hypoxemia in Miner because Miner's CWP was too small to have affected Miner's lung function, so it would not affect his arterial blood gas values. He also opined that Miner's simple CWP did not have any effect on Miner's reduced diffusion capacity because there was no description of the presence of focal emphysema, only centrilobular emphysema. Thus, Dr. Repsher

would attribute all of the reduction of Miner's diffusing capacity to cigarette smoking. Similarly, he opined that Miner's decreased FEV1 measurements were not caused by Miner' simple CWP because it was too small of an amount of lung tissue to have a measurable impact on Miner's overall lung function. Dr. Repsher concluded that Miner's death was not hastened by his simple CWP to any measurable extent because the amount of CWP was so small that its effect on lung function would be too small to measure.

Hospital Records

Dr. Paul Sheya interpreted a chest x-ray obtained on June 27, 1995 and compared it with an x-ray obtained in August 1991. (CX 5). His interpretations included mild pulmonary vascular congestion and borderline cardiomegaly, atherosclerosis and COPD, and findings suggesting right lobe consolidation that might be contributed to by underlying chronic lung disease.

Dr. King treated Miner at Castleview Hospital from June 26, 1995 through June 29, 1995 for pneumonia. (CX 5). In his admission note, Dr. King characterized Miner as a patient he had been following for COPD and Black Lung. He noted that Miner had been maintained by inhalers and was dependent on oxygen since 1989. Dr. King commented that a chest x-ray showed chronic scarring from Miner's baseline lung disease. He detected bibasilar crackles upon physical examination of Miner's lungs.

Dr. Nicholas Baldwin interpreted a chest x-ray obtained on July 1, 1996 as revealing a stable chest showing minimal hyperinflation, minimal chronic interstitial densities, and a calcified granuloma. (CX 5). Dr. Baldwin interpreted an x-ray obtained on April 23, 1998 as showing suboptimal inspiration with minimal atelectasis and/or consolidation of the left costophrenic angle, and he could not rule out small left pleural effusion.

Miner was brought to Castleview Hospital on April 21, 1998 after getting light-headed and passing out at home. (CX 5). Dr. Cameron Williams examined Miner and rendered diagnoses of syncopal episode at home, gastrointestinal hemorrhage, COPD, congestive heart failure, and senile dementia. He admitted Miner for evaluation of his blood loss and for a possible transfusion, since Miner had marginal oxygen support. Dr. Wayne Cox conducted a consultative examination, noting that Miner had a history of congestive heart failure, emphysema, and a gastrointestinal bleed a few years ago. Dr. Cox performed an upper gastrointestinal endoscopy. He detected several ulcers in Miner's stomach.

Dr. Jean-Maurice Poitras, who followed Miner while he was at Castleview Hospital in April 1998, dictated a discharge note on September 28, 1998. (CX 5). He noted that Miner had been admitted after suffering a syncopal episode with probable gastrointestinal bleed, which was confirmed by Dr. Cox. Dr. Poitras stated that Miner was treated with IV fluids and antibiotics for suspected bilateral pneumonia. He stated that Miner's family approached him regarding Miner's quality of life due to Miner's slow, inexorable downward course over the past few years. After a discussion, Dr. Poitras stated that Miner's family decided to withhold aggressive care and just to provide supportive care. Dr. Poitras stated that he allowed Miner to remain in the hospital for three days to see if there was any chance for recovery, but he determined that there was no chance. Miner was transferred to a transitional

care unit for a few days and then sent home. Dr. Poitras' discharge diagnoses were gastrointestinal bleed, congestive heart failure, COPD, and dementia.

Miner was admitted to Parkdale Care Center on May 1, 1998. (DX 106). He remained there until he expired on May 13, 1998. Dr. Potter was Miner's attending physician. He rendered admitting diagnoses of congestive heart failure, arthritis, hypertension, gastrointestinal hemorrhage, pneumonia, emphysema, dementia, and constipation. Miner was provided with physical therapy for therapeutic exercise, mobility training, and transfer training. His medications were managed. Detailed nursing notes were kept regarding Miner's condition. The following notes from May 12, 1998 were recorded:

> 2435 CNA called nurse to room, pt pale, cool to touch et non-responsive. During attempt to obtain VS pt expired. Pulse was thready et difficult to palpate, resp were shallow. Call placed to phys. Awaiting return call.
>
> 2438 spoke [with] Dr. Potter. He states probable MI. He will call family.

(DX 106).

Smoking History

Claimant testified that Miner attempted to quit smoking in 1981, and he was finally able to stop at some point in 1982. (Tr. 29). The physicians uniformly considered a smoking history in the amount of one pack of cigarettes per day beginning in 1942 and ending in 1982. Therefore, I find that Miner had a 40 pack-year smoking history.

LIVING MINER CLAIM

DISCUSSION AND APPLICABLE LAW

Mr. Kourianakis' claim was made after March 31, 1980, the effective date of Part 718, and must therefore be adjudicated under those regulations. To establish entitlement to benefits under Part 718, Claimant must establish, by a preponderance of the evidence, that he:

1. Is a miner as defined in this section; and

2. Has met the requirements for entitlement to benefits by establishing that he:

   (i) Has pneumoconiosis (see § 718.202), and

   (ii) The pneumoconiosis arose out of coal mine employment (see § 718.203), and

   (iii) Is totally disabled (see § 718.204(c)), and

   (iv) The pneumoconiosis contributes to the total disability (see § 718.204(c)); and

3. Has filed a claim for benefits in accordance with the provisions of this part.

Section 725.202(d)(1-3); *see also* §§ 718.202, 718.203, and 718.204(c).

Duplicate Claim

The provisions of § 725.309 apply to new claims that are filed more than one year after a prior denial. Section 725.309 is intended to provide claimants relief from the ordinary principles of *res judicata*, based on the premise that pneumoconiosis is a progressive and irreversible disease. *See Lukman v. Director, OWCP*, 896 F.2d 1248 (10th Cir. 1990); *Orange v. Island Creek Coal Company*, 786 F.2d 724, 727 (6th Cir. 1986); § 718.201(c) (Dec. 20, 2000). Section 725.309(d) provides that: "If the earlier miner's claim has been finally denied, the later claim shall also be denied, on the grounds of the prior denial, unless the deputy commissioner determines that there has been a material change in conditions or the later claim is a request for modification and the requirements of § 725.310 are met."

The Benefits Review Board has defined "material change in conditions" under § 725.309(d) as occurring when a claimant establishes, by a preponderance of the evidence developed subsequent to the prior denial, at least one of the elements of entitlement previously adjudicated against the claimant. *See Allen v. Mead Corp.*, 22 B.L.R. 1-61 (2000). The Board has also held that a material change in conditions may only be based upon an element which was previously denied. *Caudill v. Arch of Kentucky, Inc.*, 22 B.L.R. 1-97 (2000) (en banc on recon.) (where Administrative Law Judge found that claimant did not establish pneumoconiosis and did not specifically address total disability, the issue of total disability may not be considered in determining whether the newly submitted evidence is sufficient to establish a material change in conditions). Lay testimony alone is insufficient to establish a material change in conditions. *Madden v. Gopher Mining Co.*, 21 B.L.R. 1-122 (1999).

This matter arises under the jurisdiction of the Tenth Circuit Court of Appeals.[6] In *Wyoming Fuel Co. v. Director, OWCP*, 90 F.3d 1502 (10th Cir. 1996), the Tenth Circuit held that, in order to establish a "material change in conditions" under § 725.309, a claimant "must prove for each element that was actually decided adversely to the claimant in the prior denial that there has been a material change in that condition since the prior claim was denied. *Id.* at 1511. The Tenth Circuit further stated:

> In order to meet the claimant's threshold burden of proving a material change in a particular element, the claimant need not go as far as proving that he or she now satisfies the element. Instead, under the plain language of the statute and regulations, and consistent with res judicata, the claimant need only show that this element has worsened materially since the time of the prior denial. An example of how a claimant might show that a condition has materially worsened, the claimant might offer to compare past and present x-rays reflecting that any conditions suggesting that the claimant has pneumoconiosis have become materially more severe since the last claim was rejected. As another example, the claimant might present more extreme blood gas test results obtained since the prior denial to indicate that his or her disability has become materially

[6]Appellate jurisdiction with a federal circuit court of appeals lies in the circuit where the miner last engaged in coal mine employment, regardless of the location of the responsible operator. *Shupe v. Director, OWCP*, 12 B.L.R. 1-200 (1989)(en banc). Miner last engaged in coal mine employment in Utah.

> more severe since the last claim was rejected. However, a new interpretation of an old x-ray that was taken before the prior denial or a further blood gas result identical to results considered in the prior denial does not demonstrate that a miner's conditions has materially changed.

Id. Additionally, the Tenth Circuit stated that, if the adjudicator in the prior claim did not decide a particular entitlement issue, then there is no issue preclusion and the claimant need not demonstrate a "material change" in this element upon the filing of a subsequent claim. *Id.*

Administrative Law Judge Karst determined that Miner failed to establish the presence of pneumoconiosis as well as the presence of a totally disabling respiratory disability due to pneumoconiosis after he reviewed Miner's duplicate claim, which was filed in 1983. In his 1995 decision and order reviewing Miner's request for modification of the denial of his 1983 duplicate claim, Administrative Law Judge Roketenetz found that Miner again failed to establish pneumoconiosis or total respiratory disability due to pneumoconiosis. Within one year of Administrative Law Judge Roketenetz's decision and order, the OWCP initiated modification proceedings. Miner's 1983 duplicate living miner claim is now before the undersigned to evaluate the OWCP's modification of Administrative Law Judge Roketenetz's denial of Claimant's modification request from Administrative Law Judge Karst's denial of his 1983 duplicate claim.

Modification

Section 22 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 922, as incorporated into the Black Lung Benefits Act by 30 U.S.C. § 932(a) and as implemented by § 725.310, provides that upon his or her own initiative, or upon the request of any party on the ground of a change in conditions or because of a mistake in a determination of fact, the deputy commissioner may, at any time prior to one year after the date of the last payment of benefits, or at any time before one year after the denial of a claim, reconsider the terms of an award or a denial of benefits. § 725.310(a).

In deciding whether a mistake in fact has occurred, the United States Supreme Court stated that the Administrative Law Judge has "broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." *O'Keefe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 256 (1971).

In determining whether a change in conditions has occurred requiring modification of the prior denial, the Benefits Review Board ("Board") similarly stated that,

> the Administrative Law Judge is obligated to perform an independent assessment of the newly submitted evidence (all evidence submitted subsequent to the prior denial), considered in conjunction with the previously submitted evidence, to determine if the weight of the new evidence is sufficient to establish at least one element of entitlement which defeated entitlement in the prior decision.

Kingery v. Hunt Branch Coal Co., BRB No. 92-1418 BLA (Nov. 22, 1994); *See also Napier v. Director, OWCP*, 17 B.L.R. 1-111 (1993); *Nataloni v. Director, OWCP*, 17 B.L.R. 1-82 (1993). Furthermore,

> if the newly submitted evidence is sufficient to establish modification . . ., the Administrative Law Judge must consider all of the evidence of record to determine whether Claimant has established entitlement to benefits on the merits of the claim.

Kovac v. BNCR Mining Corp., 14 B.L.R. 1-156 (1990), *modified on recon.*, 16 B.L.R. 1-71 (1992).

Mistake in a Determination of a Fact

The OWCP did not allege that a mistake in determination of fact occurred in the prior decision and order. I have reviewed the decision and all of the evidence before Administrative Law Judge Roketenetz for consideration. Administrative Law Judge Roketenetz determined that the Director had erroneously considered Dr. Davis's 1/0 interpretation of a film dated December 12, 1990 to be a positive finding for the presence of pneumoconiosis because Dr. Davis attached a narrative report to the x-ray stating that the "small irregular opacities are not characteristic for coal workers' pneumoconiosis." Administrative Law Judge Roketenetz determined that Dr. Davis' interpretation could not constitute positive or negative evidence of pneumoconiosis. According to § 718.202(a)(1), a finding of 1/0 on a form that complies with the quality standards of § 718.102 and Appendix A to Part 718 constitutes a positive finding for the presence of pneumoconiosis. The Benefits Review Board has held that comments such as Dr. Davis' are properly analyzed under § 718.203(b) as contrary probative evidence militating against the presumption that Miner's pneumoconiosis arose out of coal mine employment. *See Cranor v. Peabody Coal Co*., 22 B.L.R. 1-1 (1999)(en banc on recon.). However, since the nine other interpretations were negative, the preponderance of the newly submitted chest x-ray evidence before Administrative Law Judge Roketenetz was still negative for the presence of pneumoconiosis. Administrative Law Judge Roketenetz also found that Dr. Shockey's June 1989 opinion attributing some element of Miner's chronic bronchitis to coal dust exposure to constitute a discrepancy with his June 1989 letter to the Miner informing Miner that Miner's chronic bronchitis and emphysema were the result of cigarette smoking and coal dust exposure. Dr. Shockey did not equivocally and unequivocally find that a causal relationship existed between Miner's respiratory disease and his coal dust exposure. However, Administrative Law Judge Roketenetz determined that he could not credit Dr. Shockey's report of pneumoconiosis as a conclusive diagnosis of pneumoconiosis, in conjunction with his finding of an discrepancy, due to the lack of other evidence supporting a finding of pneumoconiosis. Since Dr. Shockey's opinion was not properly reasoned and documented, Dr. Shockey's opinion could not have constituted a finding of pneumoconiosis under subsection (a)(4). Accordingly, Administrative Law Judge Roketenetz's finding that the evidence did not establish the presence of pneumoconiosis was correct.

Even though there were no mistaken determinations of fact in the prior decision and order that warrants a *de novo* review of the entire record, the OWCP's request for modification may still be granted if the newly submitted evidence establishes a change in conditions.

Pneumoconiosis

The OWCP may establish a change in condition by proving the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

> For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.
>
> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
>
> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

Section 718.201(a).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence. The evidence submitted after the prior decision and order consists of two chest x-ray interpretations of one chest x-ray that comply with the quality standards of § 718.102 and Appendix A to Part 718. Dr. Gotthoffer, who is a board-certified radiologist, interpreted the November 19, 1996 film as positive for pneumoconiosis. Dr. Preger, who is dually-certified as a radiologist and B-reader, also interpreted the November 19, 1996 film as positive for pneumoconiosis. There were no interpretations to the contrary. I accord probative weight enhanced by their respective credentials to the two positive interpretations rendered by Drs. Gotthoffer and Preger. I find that the November 19, 1996 x-ray is positive for the existence of pneumoconiosis. Therefore, I find that Claimant has established that Miner suffered from pneumoconiosis under subsection (a)(1).

(2) Under § 718.202(a)(2), a determination that pneumoconiosis is present may be based, in the case of a deceased miner, upon autopsy evidence. A report of an autopsy submitted in connection with a claim shall include a detailed gross macroscopic description and microscopic description of the lungs or visualized portion of the lung. § 718.106(a). A finding in an autopsy of anthracotic pigmentation shall not be sufficient, by itself, to establish the existence of pneumoconiosis. § 718.202(a)(2). Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). A diagnosis of pneumoconiosis issued by the autopsy prosector is entitled to significant probative value because the pathologist who performs the autopsy sees the entire respiratory system as well as other body systems. *See Fetterman v. Director, OWCP*, 7 B.L.R. 1-688, 1-691 (1985). It is proper to accord greater weight to the opinion of the autopsy prosector over the opinion of reviewing pathologists. *Peskie v. U.S. Steel Corp.*, 8 B.L.R. 1-126 (1985); *Similia v. Bethlehem Mines Corp.*, 7 B.L.R. 1-535 (1984). Additionally, it is reasonable to assign greater weight to physicians who have reviewed the autopsy slides over those physicians who have not. *See Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). The Board has held that anthracosis found in lymph nodes may be sufficient to establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpublished).

Three pathologists reviewed the autopsy slides. Dr. Hardy conducted an autopsy on May 13, 1998. He set forth gross macroscopic and microscopic findings, and he detailed the surgical procedure he used. His report complies with the quality standards of § 718.106. Dr. Hardy concluded that the autopsy revealed evidence of moderate simple CWP. Dr. Hardy's opinion is reasoned and documented. I find that Dr. Hardy's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist. Drs. Perper and Naeye also determined that the autopsy evidence revealed the presence of slight-moderate, simple CWP. Drs. Perper and Naeye both set forth microscopic findings and observations, and their reasoning was supported by adequate data. Their opinions are reasoned and documented. I find that the opinions of Drs. Perper and Naeye are entitled to probative weight enhanced by their credentials as board-certified pathologists. There is no dispute among the three pathologists who reviewed the autopsy evidence that Miner suffered from simple CWP. All three pathologists who reviewed the autopsy evidence are at least board-certified in clinical and anatomical pathology. Based on the credentials of the reviewing pathologists and the unanimous opinion that simple CWP is present in the autopsy slides, I find that Claimant has established the presence of pneumoconiosis through autopsy evidence under subsection (a)(2).

From the autopsy evidence, Drs. Hardy, Perper, and Naeye also diagnosed the presence of severe centrilobular emphysema. Dr. Hardy did not offer an opinion on the etiology of Miner's centrilobular emphysema. Dr. Perper found that Miner's centrilobular emphysema was causally associated to his coal mine dust inhalation, while Dr. Naeye determined that Miner's cigarette smoking was the sole cause of Miner's emphysema. Even though the presence of pneumoconiosis has already been established, it is necessary to resolve the disagreement between Dr. Perper and Dr. Naeye regarding the etiology of Miner's severe centrilobular emphysema because it impacts the reliability of their opinions on the etiology of Miner's death.

Dr. Perper determined, in his August 30, 1999 initial report of his autopsy findings, that Miner's severe centrilobular emphysema was causally associated to Miner's inhalation of coal

mine dust. He noted that Miner's cigarette smoking history cannot exclude the equally substantial contribution of significant exposure to coal mine dust containing silica. He relied upon scientific literature to support his conclusion. He also found evidence of marked focal fibrosis associated with anthracotic pigmentation (and silica crystals) due exclusively to the pneumoconiotic process since the fibrous anthracotic septa contained multiple silica crystals. Dr. Naeye criticized Dr. Perper's findings in his November 19, 1999 initial report, which he based on his autopsy findings. Dr. Naeye also referenced medical literature to support his opinion, and he identified his years of experience reviewing the lungs of miners whose dust exposures contributed to their emphysema and disability. However, he stated that Miner was not one of those miners. Dr. Perper reviewed Dr. Naeye's November 1999 report and issued a follow-up report on June 23, 2003 that addressed Dr. Naeye's criticisms. He stated that he didn't know why Dr. Naeye could not detect birefringent silica particles, since both he and Dr. Hardy detected their presence in the autopsy slides. Dr. Perper opined that Dr. Naeye minimized the severity of the CWP present in Miner's lungs. Dr. Perper stated that Dr. Naeye relied upon medical literature that is virtually obsolete. He opined that the fact that Miner stopped smoking for years before his demise reinforced the causal relationship between Miner's exposure to mixed coal dust containing silica, Miner's CWP, and Miner's COPD. Dr. Perper identified the significance of Miner's cessation of smoking followed by the appearance of opacities as reinforcing the causation of the predominant role of Miner's CWP in causing his COPD because cessation of smoking stops any progression of smoking related centrilobular emphysema, while cessation of occupational exposure does not stop the relentless process of CWP and related centrilobular emphysema. Dr. Perper set forth pathological findings and observations, and his reasoning is supported by adequate data. He also relied upon scientific literature and defended his reliance on the literature in the face of criticism from Dr. Naeye. His opinion is reasoned and documented. I find that Dr. Perper's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical, anatomical, and forensic pathologist. In a report dated June 26, 2003, Dr. Naeye responded to three specific criticisms levied by Dr. Perper. Dr. Naeye rejected a diagnosis of complicated pneumoconiosis. He reiterated his prior finding that very tiny birefringent crystals of toxic free silica that cause fibrosis and lung damage were not present in Miner's lungs. Dr. Naeye stated that he did find a few larger crystals, but they were non-toxic silicates that do not produce fibrosis or lung damage. Lastly, Dr. Naeye noted that Miner was exposed to very little, if any, mine dust, which sometimes contains toxic free silica. Instead, he stated that Miner was primarily exposed to coal dust, which is non-toxic because it contains little, if any, free silica.

Dr. Repsher rendered a consultative report and testified at the hearing regarding the etiology of Miner's centrilobular emphysema. In his June 25, 2003 report, Dr. Repsher opined that Miner suffered from COPD with severe centrilobular emphysema and severe cor pulmonale as the result of Miner's long and heavy cigarette smoking habit. Dr. Repsher elaborated on this conclusion in his testimony at the hearing. First, he testified that there is no credible, published literature that shows a person can develop clinically significant centrilobular emphysema solely from exposure to coal dust. Dr. Repsher stated that he reviewed the literature Dr. Perper referenced, and he found that Dr. Perper's opinion did not accurately reflect the findings of the articles because the articles didn't document the existence of coal dust related centrilobular emphysema and because the studies didn't control for cigarette smoking (the most likely cause of centrilobular emphysema). Dr. Repsher acknowledged that a study conducted by Cochroft

reported that coal dust contributed to the development of centrilobular emphysema after controlling for miners and non-miners who had died of ischemic heart disease and after accounting for age and smoking history. However, Dr. Repsher testified that this study was not credible and didn't actually control for cigarette smoking. Moreover, he testified that this study did not distinguish between the forms of emphysema. Dr. Repsher stated that focal emphysema is associated with coal dust exposure, but is generally not associated with any lung function abnormality. He added that centrilobular emphysema is usually associated with catastrophic impairment of the lung. Dr. Repsher then identified a report entitled "Pathology Standards for Coal Workers' Pneumoconiosis." He stated that the report identifies the standards for diagnosing pneumoconiosis by autopsy evidence. He stated that silica, which is the most toxic of the crystals, is weakly birefringent and very small and faint when viewed under a microscope. Dr. Repsher stated that Drs. Naeye and Perper both found silicate crystals that can be found in anyone. He noted that Dr. Naeye did not identify the presence of silica crystals necessary to diagnose the presence of CWP in Miner's the one centimeter lesions in Miner's lungs. Dr. Repsher added that Dr. Perper did not distinguish in his report between the weakly birefringent crystals of toxic silica and the strongly birefringent silicate crystals. He then concluded that Drs. Perper and Naeye did not find the presence of weakly birefringent silica crystals. Dr. Repsher also stated that Miner's CWP was too small to affect lung function, and he added that Miner's CWP did not have any effect on Miner's diffusion capacity because there was no evidence of focal emphysema, only centrilobular emphysema. Therefore, Dr. Repsher would attribute all of Miner's impairment to cigarette smoking.

Drs. Naeye and Repsher contend that the reliable scientific medical literature only supports the existence of a causal relationship between coal mine dust inhalation and the development of centrilobular emphysema when there is pathological evidence of the presence of toxic silica in the form of tiny, birefringent silica crystals. They argue that the larger silica crystals, called silicates, are non-toxic and more prevalent. They also argue that cigarette smoking is the most common cause of centrilobular emphysema. Drs. Naeye and Perper determined that Miner's centrilobular emphysema was not due to coal mine dust because they did not detect the presence of toxic, tiny, birefringent silica crystals. They also assert that Miner was only exposed to a limited amount of mine dust, if any at all, that could contain toxic silica crystals. Dr. Naeye stated, relying on his years of experience, that there are only a limited amount of coal seams that actually contain toxic silica crystals. He determined that condition of Miner's lungs was not consistent with the conditions of the lungs of miners who had been exposed to toxic silica crystals that led to their death based on his years of experience. Dr. Naeye's opinion is reasoned and documented. He set forth pathological and clinical findings and observations. Dr. Naeye relied upon adequate data to support his reasoning. He is board-certified in clinical and anatomical pathology, and he has extensive experience evaluating the pathology of the lungs of coal miners. He offered criticisms of Dr. Perper's report and he reaffirmed his own findings in the face of criticism from Dr. Perper. I accord probative weight to Dr. Naeye's opinions enhanced by his credentials as a board-certified pathologist. Dr. Repsher is not a pathologist, and he did not review the autopsy slides. He did review relevant medical literature, including the pathology standards for determining the presence of pneumoconiosis at autopsy. But, again, he is not a pathologist. His information regarding the condition of Miner's lungs at autopsy was based on his review of the pathological reports of Drs. Hardy, Perper, and Naeye. He set forth findings and observations, and his reasoning is supported

by adequate data. His opinion is reasoned and documented. I accord probative weight to the opinion of Dr. Repsher. However, his opinion does not carry the same degree of probative weight as do the reasoned and documented opinions of board-certified pathologists who reviewed the actual autopsy evidence.

It is difficult to resolve the inconsistencies between the assessments of Drs. Naeye and Repsher versus the assessment of Dr. Perper regarding the scientific medical literature without going beyond the scope of the undersigned's authority (i.e. not practicing medicine from the bench). Each physician levied pointed criticism at the medical literature relied upon by the other. Drs. Naeye and Repsher argue that Dr. Perper relies upon studies that did not control for cigarette smoking, which is known to be the most common cause of centrilobular emphysema. However, Dr. Repsher acknowledged that the Cochroft study claimed to have controlled for cigarette smoking. Dr. Perper argued that Dr. Naeye relied upon virtually obsolete literature. In turn, Drs. Naeye and Repsher argued that Dr. Perper failed to distinguish between tiny birefringent silica crystals that are toxic and the larger, non-toxic silicate crystals. The foregoing criticism levied by Drs. Naeye and Repsher identifies the common denominator between Dr. Perper's view on how coal mine dust causes centrilobular emphysema and the views of Drs. Naeye and Repsher. At a minimum, Drs. Perper, Naeye, and Repsher agree that the presence of tiny, birefringent silica crystals serves as a marker that coal mine dust contributed to the development of centrilobular emphysema. The specific findings of the two reviewing pathologists and the autopsy prosector must then be evaluated to determine if tiny birefringent silica crystals are present.

Drs. Naeye and Repsher argue that Miner's lungs did not contain tiny birefringent silica crystals. Dr. Naeye's opinion is based on his review of the autopsy slides and the opinions of Drs. Hardy and Perper, while Dr. Repsher's opinion is solely based on his review of the opinions of the three pathologists. Dr. Hardy, in his final note, made the following finding: "In the areas of pigment disposition polarized light microscopy showed characteristic delicate silica particles." Dr. Perper's microscopic findings from Miner's lungs included the following: "Pleura shows focal fibro-anthracosis, slight to moderate with presence of birefringent silica crystals and focal, chronic inflammatory infiltrates. In places the pleural fibrosis is severe, with subpleaural fibrosis, moderate to marked anthracosis and small numbers of birefringent silica crystals." Dr. Perper also found "[s]light to marked, scattered macules with anthracotic pigmentation in peri-vascular and peri-bronchial locations, and in the inter-alveolar septa, containing birefringent silica crystals." He also detected "[s]everal slides [sic] shows three to four adjacent, fibro-anthracotic micronodules, measuring up to 0.1 cm each. In the nodules and in the areas of anthracotic pigmentation multiple birefringent silica crystals are present. Some of the micronodules are showing surrounding scar (focal) emphysema." Dr. Perper found the presence of tiny birefringent silica crystals, Dr. Naeye did not, and Dr. Hardy found "characteristic delicate silica particles." Dr. Perper stated that Dr. Hardy detected the presence of tiny birefringent silica particles. Dr. Repsher described toxic silica as appearing weakly birefringent and very small and faint when viewed under a microscope. I infer that Dr. Hardy's finding of the "characteristic delicate silica particles" is a finding tiny birefringent silica particles, not a finding of strongly birefringent silicates. Thus, Dr. Hardy and Dr. Perper identified the presence of tiny birefringent silica crystals in Miner's lungs. Since two of the three pathologists who reviewed the autopsy slides identified the presence of tiny birefringent toxic silica crystals, I find that the

preponderance of the evidence establishes the presence of tiny birefringent toxic silica crystals. A fundamental underlying predicate of the opinions of Drs. Naeye and Repsher is the absence of tiny birefringent silica crystals. This important foundation of their opinions is contradicted by a preponderance of the evidence. Thus, I find that Dr. Perper's opinion on the etiology of Miner's centrilobular emphysema is better supported by the objective medical evidence than the opinions of Drs. Naeye and Repsher. I find that Miner's coal mine dust inhalation was a significant contributing factor to the development of Miner's centrilobular emphysema, which amounts to a finding of legal pneumoconiosis. The preponderance of the autopsy evidence supports a finding of legal pneumoconiosis in the form of severe centrilobular emphysema that was significantly contributed to by coal mine dust inhalation.

Claimant has established the presence of clinical and legal pneumoconiosis by a preponderance of the autopsy evidence under subsection (a)(2).

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. In this case, the presumption of § 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Therefore, Claimant cannot establish pneumoconiosis under subsection (a)(3).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 is set forth in subsection (a)(4) which provides in pertinent part:

> A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

§ 718.202(a)(4).

This section requires a weighing of all relevant medical evidence to ascertain whether or not the claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts, and other data on which he bases his diagnosis. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985).

Dr. King examined Miner in December 19, 1995 for the presence of pneumoconiosis. He opined that Miner probably had mixed COPD with some elements of black lung, but he did not

have proof at that time. Dr. King's report does not constitute a reasoned and documented opinion diagnosing the presence of pneumoconiosis. Therefore, his 1995 opinion cannot support a finding of pneumoconiosis. Dr. King issued a second opinion in June 2003. He reviewed the autopsy report and talked with the autopsy prosector. In his report, he determined that the autopsy prosector confirmed the presence of pneumoconiosis at the time of his death.

Dr. Guicheteau examined Miner on November 19, 1996. He provided a summary of his physical examination of Miner on that date, and after reviewing the results of a chest x-ray, PFT, ABG, and EKG, he rendered a narrative opinion on December 21, 1996. He determined that Miner suffered from simple pneumoconiosis based on the chest x-ray and Miner's history of 33 years of coal mine employment and a 40 pack-year smoking history. The Sixth Circuit Court of Appeals has held that merely restating an x-ray is not a reasoned medical judgment under § 718.202(a)(4). *Cornett v. Benham Coal, Inc.*, 227 F.3d 569 (6th Cir. 2000). The Board has also explained that, when a doctor relies solely on a chest x-ray and coal dust exposure history, a doctor's failure to explain how the duration of a miner's coal mine employment supports his diagnosis of the presence or absence of pneumoconiosis renders his opinion "merely a reading of an x-ray . . . and not a reasoned medical opinion." *Taylor v. Brown Bodgett, Inc.*, 8 B.L.R. 1-405 (1985). *See also Worhach v. Director, OWCP*, 17 B.L.R. 1-105, 1-110 (1993)(citing *Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-113 (1989)(it is permissible to discredit the opinion of a physician which amounts to no more than a restatement of the x-ray reading). Dr. Guicheteau's finding of simple pneumoconiosis was based solely on the chest x-ray interpretation rendered by Dr. Gotthoffer and Miner's smoking and coal mine employment histories. Dr. Guicheteau did not explain why Miner's smoking and coal mine employment histories were diagnostic of CWP. His opinion is not reasoned and documented. His opinion cannot support a finding of pneumoconiosis under subsection (a)(4).

Dr. Petersen interpreted a CT scan dated December 2, 1996 as showing hilar calcifications related to a pneumoconiosis such as silicosis. He also found interstitial changes that were not inconsistent with a pneumoconiosis such as anthracosis. Dr. Petersen did not provide an opinion on the etiology of his findings of silicosis and anthracosis. Therefore, they do not amount to findings of clinical pneumoconiosis under § 718.201(a)(1). Dr. Petersen's CT scan interpretation cannot support a finding of pneumoconiosis under subsection (a)(4).

Dr. Hill issued a narrative report on December 4, 1996. He opined that Miner's coal dust exposure contributed to some degree to his pulmonary limitation based on Miner's appropriate exposure history, the December 2, 1996 CT scan, and scientific literature. Dr. Hill also attributed some of Miner's FEV1 limitation to Miner's coal dust exposure. He noted that Miner's smoking history clearly significantly contributed to Miner's pulmonary limitation, and he added that there is reason to believe that there is some contribution from coal dust exposure. Dr. Hill's opinion does not constitute a diagnosis of legal or clinical pneumoconiosis. In order for the chronic dust disease to be defined as "arising out of coal mine employment", the Act requires that the chronic pulmonary or respiratory impairment be significantly related to or substantially aggravated by dust exposure in coal mine employment. § 718.201(b). Dr. Hill found that Miner's smoking history was a significant contributing cause of Miner's pulmonary impairment, but he only found that Miner's coal dust exposure contributed "some" to his pulmonary impairment. Dr. Hill stated that he was unable to provide an exact percentage with

regard to the amount that coal dust exposure contributed. Thus, I find that Dr. Hill's opinion does not amount to a diagnosis of legal pneumoconiosis. His opinion cannot support a finding of pneumoconiosis under subsection (a)(4).

Dr. Hardy did review some of Miner's medical records, but his opinion regarding the existence of pneumoconiosis was based solely on the autopsy evidence. Thus, I will not consider his opinion under subsection (a)(4).

Dr. Perper rendered a narrative opinion on August 30, 1999. He reviewed Miner's medical records and the autopsy slides. Dr. Perper opined that Miner suffered from slight-moderate simple CWP based on a sufficient history of exposure, respiratory symptomatology, severe objective respiratory dysfunction based on the PFTs and ABGs, clinical findings of barrel shaped chest and shortness of breath, as well as autopsy evidence of CWP. Dr. Perper considered an accurate account of Miner's smoking and coal mine employment history. He set forth clinical and pathological findings and observations, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Perper's opinion is entitled to probative weight enhanced by his credentials as a board-certified pathologist.

Dr. Naeye found that the autopsy evidence established the presence of simple CWP, severe centrilobular emphysema, and chronic bronchitis. He commented that the absence of radiological evidence of CWP was indicative of the mildness of Miner's CWP. Dr. Naeye opined that Miner's clinical, arterial blood gas and pulmonary function abnormalities were the consequence of Miner's 40 pack years of cigarette smoking. Dr. Naeye concluded that Miner's centrilobular emphysema and COPD was due solely to cigarette smoking. He referenced scientific medical literature that found the effects of cigarette smoking to be three times that of coal mine dust for miners who worked underground and away from the coal face. Dr. Naeye set forth clinical and pathological findings and observations. His reasoning is supported by adequate data. He considered an accurate account of Miner's smoking and coal mine employment history. Dr. Naeye relied upon scientific literature to support his conclusions and he defended his opinion in the face of criticism. His opinion is reasoned and documented. I find that Dr. Naeye's opinion is entitled to probative weight enhanced by his credentials as a board-certified pathologist.

Dr. Repsher opined that there was no radiographic evidence of CWP, but he did find mild, histological simple CWP. He attributed Miner's COPD with severe centrilobular emphysema and cor pulmonale to Miner's smoking history. Dr. Repsher relied upon scientific medical literature to support his conclusions. He set forth clinical observations and findings, and his reasoning is supported by adequate data. Dr. Repsher considered and accurate account of Miner's smoking and coal mine employment history. His opinion is reasoned and documented. I find that Dr. Repsher's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

The physicians who reviewed Miner's medical records, as well as the autopsy evidence which is the most reliable, determined that Miner suffered from simple CWP. Based on the opinions of Drs. Naeye, Perper, King and Repsher, I find that Claimant established that Miner suffered from pneumoconiosis by a preponderance of the evidence under subsection (a)(4).

I have determined that Claimant established the presence of pneumoconiosis under subsections (a)(1), (a)(2), and (a)(4). Therefore, I find that Claimant has established that Miner suffered from pneumoconiosis at the time of his death.

Arising out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must also prove that pneumoconiosis arose, at least in part, out of his coal mine employment. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment. *Id.* I have found that Claimant has established at least 30 years of coal mine employment. Dr. Repsher offered rebuttal evidence when he opined that Dr. Preger's 1/0 t, t x-ray interpretation was not consistent with a finding of pneumoconiosis arising out of coal mine employment. Rather, since t opacities are lineal opacities and coal workers' pneumoconiosis produces rounded p, q, or r shaped opacities, Dr. Repsher found that Dr. Preger's April 9, 1997 interpretation identified findings consistent with asbestosis and the classic changes of cigarette smoking. The autopsy evidence confirms the presence of classic changes due to cigarette smoking, but there were no findings identifying the presence of asbestosis. The record does not contain any evidence that Miner was exposed to asbestos. Every physician who reviewed the autopsy evidence found that presence of at least mild histological coal workers' pneumoconiosis. I find that Dr. Repsher's opinion is insufficient to rebut the presumption that Miner's clinical and legal pneumoconiosis arose out of his 30 or more years of coal mine employment. Therefore, I find that Claimant's pneumoconiosis arose out of his coal mine employment in accordance with the rebuttable presumption set forth in § 718.203(b).

Total Disability

To prevail, Claimant must also demonstrate that he is totally disabled from performing his usual coal mine work or comparable work due to pneumoconiosis under one of the five standards of § 718.204(b) or the irrebuttable presumption referred to in § 718.204(b). The Board has held that under Section 718.204(b), all relevant probative evidence, both "like" and "unlike" must be weighed together, regardless of the category or type, in the determination of whether the Claimant is totally disabled. *Shedlock v. Bethlehem Mines Corp.*, 9 B.L.R. 1-195 (1986); *Rafferty v. Jones & Laughlin Steel Corp.*, 9 B.L.R. 1-231 (1987). Claimant must establish this element of entitlement by a preponderance of the evidence. *Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986).

Employer stipulated that Miner was totally disabled due to a respiratory impairment. Therefore, the remaining issue for determination is whether Miner's totally disabling respiratory impairment was due, at least in part, to Miner's pneumoconiosis arising out of coal mine employment.

Total Disability Due to Pneumoconiosis

The amended regulations at § 718.204(c) contain the standard for determining whether Miner's total disability was caused by Miner's pneumoconiosis. Section 718.204(c)(1)

determines that a miner is totally disabled due to pneumoconiosis if pneumoconiosis, as defined in § 718.201, is a "substantially contributing cause" of the miner's totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a "substantially contributing cause" of the miner's disability if it has a material adverse effect on the miner's respiratory or pulmonary condition or if it materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. §§ 718.204(c)(1)(i) and (ii). Section 718.204(c)(2) states that, except as provided in § 718.305 and § 718.204(b)(2)(iii), proof that the Miner suffered from a totally disabling respiratory or pulmonary impairment as defined by §§ 718.204(b)(2)(i), (ii), (iv), and (d) shall not, by itself, be sufficient to establish that the miner's impairment was due to pneumoconiosis.

Except as provided by § 718.204(d), the cause or causes of a miner's total disability shall be established by means of a physician's documented and reasoned medical report. § 718.204(c)(2). The Sixth Circuit Court of Appeals has stated that pneumoconiosis must be more than a "de minimus or infinitesimal contribution" to the miner's total disability. *Peabody Coal Co. v. Smith,* 12 F. 3d 504, 506-507 (6th Cir. 1997). The Sixth Circuit has also held that a claimant must affirmatively establish only that his totally disabling respiratory impairment (as found under § 718.204) was due 'at least in part' to his pneumoconiosis. *Cf*. 20 C.F.R. 718.203(a)." *Adams v. Director, OWCP*, 886 F.2d 818, 825 (6th Cir. 1988); *Cross Mountain Coal Co. v. Ward*, 93 F.3d 211, 218 (6th Cir. 1996)(opinion that miner's "impairment is due to his combined dust exposure, coal workers' pneumoconiosis as well as his cigarette smoking history" is sufficient). More recently, in interpreting the amended provision at § 718.204(c), the Sixth Circuit determined that entitlement is not precluded by "the mere fact that a non-coal dust related respiratory disease would have left the miner totally disabled even without exposure to coal dust." *Tennessee Consolidated Coal Co. v. Director, OWCP [Kirk}*, 264 F.3d 602 (6th Cir. 2001). A miner "may nonetheless possess a compensable injury if his pneumoconiosis materially worsens this condition." *Id*.

The reasoned medical opinions of those physicians who diagnosed the existence of pneumoconiosis and who found that Miner was totally disabled are more reliable for assessing the etiology of Miner's total disability. *See, e.g. Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819 (4th Cir. 1995); *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109 (4th Cir. 1995).

Dr. Badger's 1995 opinion does not address Miner's pulmonary capacity, nor its etiology, nor does the 1995 opinion of Dr. King or the 1996 opinions of Drs. Guicheteau and McCellan. In his December 1996 opinion, Dr. Hill found that Miner's severe pulmonary limitation had been significantly contributed to by his 40 pack-year smoking history, and to some degree by Miner's coal dust exposure based on Miner's history of exposure to coal dust, a December 1996 CT scan of Miner's chest, and medical literature that suggests that the effects of mine-dust exposure are one-third as severe as cigarette smoking. Dr. Hill stated that he could not identify the exact percentage that Miner's coal dust exposure contributed to his pulmonary limitation. He conducted a physical examination, reviewed the findings of objective testing, and he considered an accurate account of Miner's smoking and coal mine employment history. Dr. Hill set forth clinical observations and findings, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Hill's opinion is entitled to probative weight.

In December 1996, Dr. Guicheteau opined that Miner was totally disabled due to simple pneumoconiosis and his history of cigarette smoking. He stated that his opinion was based on the results of his physical examination, Miner's history, the chest x-ray, PFT, and ABG. He opined that Miner's smoking history does not negate the aspect of 33 years of coal dust exposure contributing to Miner's disability. Dr. Guicheteau then concluded that Miner's simple pneumoconiosis was a presumptive cause of his disability, adding that a biopsy would be the ideal method of diagnosing the etiology. However, he noted that Miner could not undergo a biopsy because of the intrinsic risks. He added that it is difficult to prove that pneumoconiosis is not a contributing factor without biopsy evidence. Dr. Guicheteau examined Miner, reviewed the findings of objective testing, and he considered an accurate account of Miner's smoking and coal mine employment history. He set forth clinical observations and findings, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Guicheteau's opinion is entitled to probative weight.

Dr. Hardy, the autopsy prosector, did not render an opinion regarding Miner's pulmonary capacity prior to death nor its etiology.

In his August 30, 1999 opinion, Dr. Perper did not render an opinion regarding Miner's pulmonary capacity at the time of his death nor its etiology. In his June 2003 supplemental opinion, Dr. Perper opined that Miner's CWP, based on its association with Miner's hypoxemia and the causal association between coal dust and centrilobular emphysema, establish that Miner's totally disabling respiratory disability was contributed to by his CWP. He noted that the fact that Miner stopped smoking for years before his demise reinforces the predominant role of Miner's CWP in the causation of his significant COPD because cessation of smoking stops the progression of centrilobular emphysema while cessation of occupational exposure does not stop the relentless process of CWP and related centrilobular emphysema because of the toxic effects of the mixed coal dust entrapped in the lungs. Dr. Perper reviewed Miner's autopsy slides and medical records, he relied upon scientific medical literature, and he considered an accurate account of Miner's smoking and coal mine employment histories. He set forth clinical and pathological findings and observations, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Perper's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical, anatomical, and forensic pathologist.

Dr. Naeye, in his November 1999 opinion, found that Miner suffered from simple CWP based on the autopsy evidence. He concluded that Miner's CWP did not produce any disability during Miner's lifetime due to the mild nature of the CWP. Dr. Naeye determined that Miner was totally disabled in his late years by a combination of severe centrilobular emphysema, chronic bronchitis, cardiac failure, and dementia. He did not consider Miner's severe centrilobular emphysema to constitute legal pneumoconiosis, which is contrary to the undersigned's determination that a preponderance of the evidence establishes that Miner's coal dust exposure contributed to Miner's centrilobular emphysema. In his supplemental June 2003 opinion, Dr. Naeye did not directly address the etiology of Miner's totally disabling respiratory impairment. He did cite a scientific study that supported his citation to Dr. Perper's opinion, which he characterized as "[t]he clinical, arterial blood gas and pulmonary function abnormalities to which Dr. Perper refers were the consequence of 40 pack years of cigarette

smoking." Beyond, that comment, Dr. Naeye did not address the etiology of Miner's totally disabling respiratory impairment in his June 2003 opinion. Dr. Naeye reviewed Miner's autopsy slides and medical records. He set forth clinical and pathological findings and observations. He did find that Miner suffered from simple clinical CWP. However, his opinion that Miner's severe centrilobular emphysema was not due, at least in part to coal dust exposure, is not supported by the objective evidence and it is contrary to the undersigned's determination that Miner's coal dust exposure did in fact contribute to his centrilobular emphysema. Therefore, I find that Dr. Naeye's opinion does not constitute a reasoned and documented opinion for determining the etiology of Miner's totally disabling respiratory impairment. I accord Dr. Naeye's opinion a lesser degree of probative weight for the purposes of the analysis conducted under § 718.204(c).

Dr. King, in his June 2003 opinion, opined that the main cause of Miner's disability and death was his emphysema and pulmonary hypertension, to which CWP served as a contributory factory. He stated that Miner's main disability of emphysema and pulmonary hypertension was mostly caused by cigarette smoking, but added that Miner's coal dust exposure was a significant contributing factor. Dr. King noted that Miner clinical pneumoconiosis cause nodule formation that altered Miner's lung architecture and contributed to Miner's hypoxemia. He then found that Miner's hypoxemia and altered lung architecture led to pulmonary hypertension, which in turn damaged Miner's heart. Dr. King treated Miner for six years from 1991 through 1997. He reviewed his office notes and the neurological, cardiac, and pulmonary consultative opinions that were submitted to him while he was Miner's treating physician. He also reviewed Dr. Hardy's autopsy report and spoke with Dr. Hardy. Dr. King submitted Miner to physical examinations, reviewed the results of objective testing and consultative examinations by other physicians, and he considered an accurate account of Miner's coal dust exposure and smoking history. He set forth clinical observations and findings, and his reasoning is supported by adequate data. His opinion is reasoned and documented. In determining the weight to be accorded to a treating physician's opinion, the amended regulations at § 718.104(d) (2002) are not directly applicable because this evidence was developed prior to January 19, 2001, but it is instructive. *See Wolf Creek Collieries v. Director, OWCP [Stephens]*, 298 F.3d 511 (6th Cir. 2002). An administrative law judge may rely upon the well-reasoned and well-documented opinion of a treating physician as substantial evidence in awarding that physician's opinion controlling weight based upon four factors: (1) nature of relationship; (2) duration of relationship; (3) frequency of treatment; and (4) extent of treatment. § 718.104(d) (2002). Dr. King treated Miner for six years, examining him four to six times per year. He attended to Miner when Miner was hospitalized in 1995. I find that Dr. King obtained superior and relevant information during the six years he served as Miner's treating physician from which to base an opinion regarding the etiology of Miner's total disability. Since Dr. King's opinion is reasoned and documented and he is board-certified in internal medicine, based on the quality and quantity of the information he obtained during his six year tenure as Miner's treating physician, I accord substantial probative weight to Dr. King's opinion.

Dr. Repsher rendered a narrative opinion in June 2003. He found, among other diagnoses, that Miner had mild CWP that was probably of no clinical significance, COPD with severe centrilobular emphysema and severe cor pulmonale probably as a result of Miner's long and heavy cigarette smoking history. He opined that Miner did not suffer during his life from the

mild histological simple CWP because Miner only had very mild histological CWP that was not radiographically visible, Miner's death certificate did not even mention CWP, and because Dr. Perper and Dr. Naeye agreed that Miner only had slight or moderate histological CWP. He added that the amount of overall lung tissue involved with Miner's simple CWP was so small that it would not be anticipated to significantly affect Miner's overall lung function, which meant that it could not have affected Miner's lung function during his lifetime. Dr. Repsher testified at the hearing in July 2003. He reiterated his findings that Miner suffered from insignificant lung pathology and very severe COPD that was complicated by centrilobular emphysema and cor pulmonale. Dr. Repsher testified that Miner's major medical problem was COPD, which forced him to retire and caused his disability during his lifetime. He reiterated his opinion that Miner's COPD, centrilobular emphysema, and cor pulmonale were caused by Miner's smoking history and not his coal dust exposure because the pathological descriptions of Miner's CWP showed that it only involved a very small fraction of Miner's lungs. Thus, it could not have caused any measurable impact on Miner's lung function. He also testified that Miner's simple histological CWP was not a contributing cause to Miner's severe heart disease or cor pulmonale, adding that heredity and cigarette smoking are the most common causes of the type of heart disease Miner had. Dr. Repsher acknowledged that he did not know that the physician who signed Miner's death certificate had not reviewed the autopsy findings when he issued his June 2003 report. He also acknowledged that he only reviewed Dr. Perper's summary of the medical records for his June 2003 report; he didn't review the actual medical records. He testified that Miner's simple CWP did not cause any hypoxemia because it was too small to have affected his lung function. Dr. Repsher opined that Miner's CWP did not have any effect on Miner's reduced diffusing capacity because there was no description of focal emphysema. Therefore, he attributed all of Miner's reduction in diffusing capacity to Miner's smoking history. He also found that Miner's decreased FEV1 values were not related to Miner's simple CWP because Miner's CWP was too small to have any measurable impact on Miner's lungs. Dr. Repsher set forth clinical observations and findings. He reviewed a summary of Miner's medical records and he considered an accurate account of Miner's smoking and coal mine employment history. However, his opinion is predicated, in part, on the finding that Miner's severe centrilobular emphysema was solely related to cigarette smoking. That predicate is contrary to the preponderance of the medical opinion evidence and the undersigned's determination that Miner's severe centrilobular emphysema was due, at least in part, to his coal dust exposure. Dr. Perper identified the presence of focal emphysema, which undercuts Dr. Repsher's testimony regarding the etiology of Miner's reduced diffusion capacity. He acknowledged that his partial reliance on the absence of CWP as a cause of death on the death certificate was misplaced because the autopsy reports were not available to the physician who signed the death certificate. The undersigned determined that the newly submitted radiographic evidence supports a finding of pneumoconiosis, but Dr. Repsher relied on the absence of radiographic evidence of CWP to support his opinion on the minimal nature of Miner's CWP. For these reasons, I find that Dr. Repsher's opinion is not reasoned and documented. I accord Dr. Repsher's opinion a lesser degree of probative weight.

Based on the opinions of Drs. Hill, Guicheteau, King, and Perper, I find that Miner's totally disabling respiratory impairment was due, at least in part, to Miner's pneumoconiosis arising out of coal mine employment. Drs. Naeye and Repsher offered opinions to the contrary. They found that Miner suffered from mild histological CWP that was to clinically insignificant

to cause any impairment during Miner's lifetime. This portion of their opinion is supported by the objective medical evidence based on the minimal nature of Miner's clinical pneumoconiosis at autopsy. However, their opinions did not consider the impact of Miner's legal pneumoconiosis in the form of severe centrilobular emphysema due to coal dust exposure. Drs. Naeye and Repsher found that Miner's severe COPD, which consisted of severe centrilobular emphysema and cor pulmonale, was due to Miner's smoking history. Their opinions support a finding regarding the significance of the role of Miner's severe centrilobular emphysema in creating Miner's totally disabling respiratory impairment. Their failure to consider the role of Miner's legal pneumoconiosis renders their opinions less probative than the opinion of Dr. Perper. Therefore, I find that Miner's totally disabling respiratory impairment was due, at least in part, to Miner's clinical and legal pneumoconiosis arising out of coal mine employment.

Claimant has established, through the newly submitted evidence, that Miner suffered from a totally disabling respiratory impairment due, at least in part, to his clinical and legal pneumoconiosis arising out of coal mine employment. Miner's duplicate claim was previously denied on the basis that Miner did not establish the presence of pneumoconiosis or that he was totally disabled due to pneumoconiosis. This newly submitted evidence establishes a change in condition since the prior denial of benefits that would warrant modification of the previous denial of Miner's duplicate claim. Since the newly submitted evidence also establishes a material change in condition regarding the two conditions of entitlement previously adjudicated against Miner, it also establishes a material change in condition under § 725.309. Miner's duplicate claim must not be denied on the basis of the prior denial of his initial claim. Rather, the entire record must be reviewed *de novo* to determine if Claimant should be awarded benefits for the living miner claim filed by Miner.

DE NOVO REVIEW OF ENTIRE RECORD

The newly submitted medical evidence contained hospital records and medical opinions based on chest x-rays, a CT scan, pulmonary function tests, arterial blood gas studies, autopsy evidence developed between 1995 and Miner's death on May 13, 1998.

Administrative Law Judge Karst considered the following evidence in the decision and order denying benefits rendered on August 4, 1986: (1) nine negative interpretations and one positive interpretation of six chest x-rays; (2) five pulmonary function tests from 1980, 1983, 1985, and 1986, from which 2 pre-bronchodilator tests (10/31/83 and 4/2/86) meet the regulatory criteria for total disability; (3) eight arterial blood gas studies from 1980, 1981, 1983, 1985, and 1986, of which three of the five studies meet the regulatory criteria for total disability; (4) 1980 examination report from Dr. Shockey concluding that Miner suffered from chronic bronchitis, mild COPD, severe hypoxemia, right carotid bruit, and possible pulmonary artery hypertension, all of which were possibly related to coal dust exposure; (5) 1983 examination report of Dr. Bekemeyer concluding that Miner suffered from carotid bruit and chronic bronchitis unrelated to coal dust exposure, but added that an industrial bronchitis component may be contributing to Miner's condition; (6) 1983 examination report of Dr. Farney concluding that Miner suffered from chronic bronchitis and emphysema secondary to cigarette smoking, and he ruled out a diagnosis of CWP; (7) 1984 examination report of Dr. Repsher finding no evidence of CWP

based on a negative chest x-ray, the obstructive and not restrictive defect found by PFT, and due to Miner's long smoking history, but concluding that Miner has COPD likely due to cigarette smoking, probable mild bronchial asthma, varying hypoxemia probably due to COPD and bronchial asthma, probable organic heart disease of unknown etiology, and peripheral vascular disease shown by right carotid bruit; (8) 1985 report of Dr. Rasmussen reviewing five PFTs, from which he concluded that Miner had a moderately severe pulmonary insufficiency that renders Miner totally disabled from performing his previous coal mine employment; (9) 1986 examination report of Dr. Farney concluding that Miner suffered from a moderately severe obstructive airways disease due to chronic bronchitis and emphysema secondary to cigarette smoking, and he also opined that Miner was not totally disabled from performing his previous coal mine employment and that there was no evidence of pneumoconiosis; and (10) Dr. Farney's testimony at the hearing reiterating the findings and conclusions contained in his 1986 examination report.

In the decision and order rendered by Administrative Law Judge Roketenetz on November 30, 1995, he considered the following medical evidence developed since the prior denial of benefits on August 4, 1986 as well as the evidence that was developed in the prior claim: (1) nine negative interpretations and one positive interpretation of nine chest x-rays films;[7] (2) the 1983, 1990, and 1995 examination reports of Dr. Farney, and Dr. Farney's testimony at the hearing in 1995 which included his finding that Miner did not suffer from pneumoconiosis but demonstrated respiratory disabilities characteristic of those found in cigarette smokers, such as chronic bronchitis; (3) Dr. Repsher's 1984 examination report finding that Miner suffered from mild COPD likely due to cigarette smoking, and also finding that Miner did not suffer from pneumoconiosis; (4) the 1990 examination reports of Drs. Lincoln and Madsen diagnosing COPD but not attributing it to coal dust exposure; (5) the diagnoses of Drs. Bekemeyer, Rasmussen, and Morgan diagnosing respiratory problems but not attributing them to coal dust exposure; (6) the numerous examination reports of Dr. Collins from 1989 through 1990 finding that Miner suffered from severe COPD, but never attributing it to coal dust exposure, which was followed by a 1990 letter stating that Miner had CWP in part based on the absence of a significant smoking history; (7) the 1980 examination report of Dr. Shockey finding that Miner exhibited symptoms of chronic bronchitis but not finding evidence of CWP based on a negative chest x-ray; and (8) the 1989 examination report of Dr. Shockey finding that Miner may have had some element of chronic bronchitis resulting from his dust exposure, as well as a 1989 letter from Dr. Shockey to Miner indicating that Miner's chronic bronchitis and emphysema were the result of coal dust exposure and cigarette smoking; and the 1992 report of Dr. King that he had followed Miner's medical treatment since 1991 and his statement that he believed that Miner suffered from COPD and black lung.

The evidence developed through 1995 supports a finding that Miner suffered from a totally disabling respiratory impairment related to COPD, chronic bronchitis, and emphysema. The preponderance of the evidence developed through 1995 did not establish the presence of

[7] In the November 30, 1995 decision and order, Administrative Law Judge Roketenetz determined that the Director had erroneously considered Dr. Davis's 1/0 interpretation of a film dated December 12, 1990 to be a positive finding for the presence of pneumoconiosis because Dr. Davis attached a narrative report to the x-ray stating that the "small irregular opacities are not characteristic for coal workers' pneumoconiosis." Administrative Law Judge Roketenetz determined that Dr. Davis' interpretation could not constitute positive or negative evidence of pneumoconiosis.

pneumoconiosis. Miner died on May 13, 1998 and an autopsy was conducted that day. The preponderance of the autopsy evidence demonstrated that Miner suffered from simple clinical pneumoconiosis, as well as from legal pneumoconiosis in the form of severe centrilobular emphysema that was significantly contributed to by Miner's coal dust exposure and cigarette smoking. The physicians who had the benefit of reviewing Miner's autopsy report and the autopsy slides unanimously determined that Miner at least suffered from mild simple clinical pneumoconiosis arising out of coal mine employment. The preponderance of the pathology evidence also established that Miner's severe centrilobular emphysema was significantly contributed to by Miner's coal dust exposure, which constitutes a diagnosis of legal pneumoconiosis. I accord greater probative weight to the more recent medical opinions that were based on the findings derived from Miner's autopsy based on the opinions of the physicians of record who determined that autopsy evidence is the most reliable evidence for determining the presence or absence of pneumoconiosis. After reviewing all of the evidence, I find that Claimant has established that Miner suffered from pneumoconiosis arising out of coal mine employment at the time of his death under § 718.202(a)(2) and (a)(4). After reviewing the entire record and based on the parties' stipulation that Miner engaged in at least 30 years of coal mine employment, I continue to hold that there is insufficient evidence to rebut the presumption that Miner's pneumoconiosis arose out of his coal mine employment in accordance with § 718.203(b). The parties stipulated that Miner was totally disabled due to a respiratory impairment at the time of his death. That stipulation is supported by the medical evidence contained in the record. Therefore, I find that Miner suffered from a totally disabling respiratory impairment under § 718.204(b)(2). The previously submitted evidence considered by Administrative Law Judges Karst and Roketenetz contained several findings that Miner suffered from a moderate-to-severe respiratory impairment related to Miner's COPD, chronic bronchitis, and emphysema. Most of those opinions found Miner's cigarette smoking to be the etiology of Miner's respiratory impairment. However, the preponderance of the autopsy evidence established that Miner's centrilobular emphysema, which was severe by the time of his death, was significantly contributed to by Miner's coal dust exposure. I find that the preponderance of the evidence establishes that Miner's totally disabling respiratory impairment was due, at least in part, to his pneumoconiosis arising out of coal mine employment. I find that Miner was entitled to benefits under the Act at the time of his death.

Entitlement

Claimant, Mary Kourianakis, has proven by a preponderance of the evidence, that Emmanuel Kourianakis suffered from a totally disabling respiratory impairment due, at least in part, to pneumoconiosis arising out of coal mine employment. Therefore, Mrs. Kourianakis is entitled to receive benefits under the Act on behalf of Mr. Kourianakis. Benefits are awarded to Mrs. Kourianakis on behalf of Mr. Kourianakis pursuant to a request for modification under § 725.310 filed by the District Director, Office of Workers' Compensation Programs. The undersigned granted the request for modification based on a change in conditions as established by the newly submitted evidence. The undersigned cannot determine from the evidence the month of onset of Miner's totally disabling respiratory impairment due, at least in part, to pneumoconiosis arising out of coal mine employment. Therefore, benefits are payable to Mrs. Kourianakis beginning with April 1997, which is the month in which the District Director, Office of Workers' Compensation Programs filed a request for modification. *See* § 725.503(d)(2).

SURVIVOR CLAIM

DISCUSSION AND APPLICABLE LAW

Mrs. Kourianakis filed her survivor's claim on July 28, 1998. Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). The Act provides that benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). In order to receive benefits, the claimant must prove that:

1). The miner had pneumoconiosis;

2). The miner's pneumoconiosis arose out of coal mine employment; and

3). The miner's death was due to pneumoconiosis.

§§ 718.205(a). Failure to establish any of these elements by a preponderance of the evidence precludes entitlement. *See Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987).

Pneumoconiosis

In establishing entitlement to benefits, Claimant must initially prove the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). The undersigned has already determined that Claimant has established the presence of pneumoconiosis arising out of coal mine employment based on the autopsy evidence and the narrative medical opinions of those physicians who reviewed evidence derived from Miner's autopsy. The undersigned also determined that the presumption that Miner's pneumoconiosis arose out of his coal mine employment was not rebutted under § 718.203(b). I incorporate the findings of pneumoconiosis arising out of coal mine employment, which were rendered in claim of the deceased miner, into the analysis of Claimant's claim for survivor benefits because the evidence is identical. Therefore, I find that Claimant has established that Miner suffered from pneumoconiosis arising out of coal mine employment. Thus, in order to be entitled to benefits, Claimant must now establish that Miner' death was due to pneumoconiosis.

Death Due to Pneumoconiosis

Mrs. Kourianakis has established, by a preponderance of the evidence, that Miner suffered from pneumoconiosis arising out of coal mine employment. She must now prove that Miner's death was due to pneumoconiosis in order to be entitled to benefits. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that an eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that pneumoconiosis was the cause of the Miner's death, or

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where death was caused by complications of pneumoconiosis, or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. § 718.205(c)(5). The presumption set forth in § 718.304 is not applicable because Claimant has not established the presence of complicated pneumoconiosis. Therefore, in order for Claimant to be entitled to benefits, she must show that pneumoconiosis was the direct cause of Miner's death or that pneumoconiosis hastened Miner's death.

A death certificate, in and of itself, is an unreliable report of the miner's condition and it is error for an administrative law judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. *Smith v. Camco Mining, Inc.*. 13 B.L.R. 1-17 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988). Dr. Potter signed Miner's death certificate May 13, 1998. He identified the immediate cause of Miner's death as cardiac arrest, with underlying causes of cardiomegaly, chronic cor pulmonale, and COPD. Dr. Potter had acted as Miner's attending physician from May 1, 1998 to the time of Miner's death at the Parkdale Care Center. Dr. Potter noted diagnoses of congestive heart failure, arthritis, hypertension, gastrointestinal hemorrhage, pneumonia, emphysema, dementia, and constipation. He managed Miner's medications. His initial opinion, upon being informed of Miner's death by the nurse on duty, was that Miner probably suffered a myocardial infarction. Dr. Potter possessed personal knowledge of Miner from which to assess the cause of death. However, he did not review the results of the autopsy before signing the death certificate. Since Dr. Potter had sufficient knowledge of Miner from which to assess the cause of death, I find that the death certificate is entitled to probative weight. However, the opinions of the physicians who reviewed the results of the autopsy are more probative of the cause of Miner's death.

Dr. Poitras, who followed Miner while he was hospitalized at Castleview Hospital in April 1998, until he was transferred to the Parkdale Care Center, and dictated a discharge note on September 28, 1998. Miner had been admitted to the hospital on April 21, 1998 after suffering a syncopal episode at home. Dr. Poitras noted that Dr. Cox had confirmed the presence of a gastrointestinal bleed that was the likely cause of Miner's syncopal episode. Dr. Poitras treated Miner with IV fluids and antibiotics for a suspected pneumonia. After Miner's family decided to withhold aggressive care, Dr. Poitras allowed Miner to stay in the hospital for three days to see if there was a chance for recovery. He determined that there was no chance for Miner to recover, noting that Miner had undergone an inexorable downhill decline over the past few years. His

diagnoses on discharge were gastrointestinal bleed, congestive heart failure, COPD, and dementia. While Dr. Poitras was not attending to Miner when he died and he did not render an opinion regarding the cause of Miner's death, his opinion is probative of Miner's respiratory capacity in the weeks immediately preceding Miner's death.

Dr. Hardy conducted the autopsy on May 13, 1998. He rendered final anatomic findings from his review of Miner's lung, concluding that Miner suffered from moderate simple CWP, anthracosilicosis, mild pleural adhesions, severe pulmonary hypertension, severe centrilobular emphysema, diffuse alveolar damage, organizing pneumonia, and focal bronchiolitis obliterans at the time of his death. From his review of Miner's heart, he determined that Miner was suffering from cardiomegaly, severe cor pulmonale in the form of right ventricular hypertrophy, left ventricular hypertrophy, moderate ischemic heart disease, mild mitral valve calcifications, mild-moderate arteriosclerosis, and a history of hypertension. Dr. Hardy concluded that dysrhythmia secondary to ischemic heart disease was the possible underlying cause of Miner's death. However, in the body of his final note, Dr. Hardy stated that he was unable to determine the immediate underlying cause of Miner's death. He opined dysrhythmia was the likely underlying cause given the degree of Miner's ischemic heart disease, but he could not exclude the possibility of an intracranial process or another etiology such as a gastric/colonic bleed. Dr. Hardy considered an accurate account of Miner's smoking and coal dust exposure history and he reviewed records from Miner's final hospitalization at Castleview Hospital in April 1998. He set forth pathological findings and observations, and his reasoning is supported by adequate data. His opinion is reasoned and documented and he is board-certified in clinical and anatomic pathology. However, I accord a limited degree of probative weight to his opinion since he opined that he could not definitively determine the cause of Miner's death; he only offered an opinion on the likely underlying cause of death and could not exclude other processes.

Dr. Perper issued an initial opinion in August 1999. He found that Miner suffered from slight-moderate clinical pneumoconiosis and moderate-marked centrilobular emphysema that was significantly contributed to by Miner's coal dust exposure. Dr. Perper also found that Miner suffered from severe cor pulmonale and arteriosclerosis consistent with a finding of severe pulmonary hypertension. Dr. Perper opined that Miner's CWP was a substantial contributory cause of Miner's death based on the significant presence of CWP and associated centrilobular emphysema. He noted that the marked focal fibrosis associated with anthracotic pigmentation and the silica crystals was clearly due exclusively to the pneumoconiotic process, since the fibrous anthracotic septa contained multiple silica crystals. He found that the most likely mechanism of death by which pneumoconiosis contributed to death was by hypoxemia that triggered or aggravated an arrhythmia caused by Miner's ischemic heart disease. Dr. Perper stressed that Miner's coronary arteriosclerotic heart disease, which was slight-moderate, was overshadowed by Miner's pulmonary disease. Dr. Perper then concluded that the primary cause of Miner's death was a result of the combined heart disease with cardiomegaly and severe COPD with simple CWP. Dr. Perper rendered a supplemental report in June 2003 in response to criticisms leveled by Dr. Naeye. He reviewed and summarized additional medical records, including Miner's hospitalization records from Castleview Hospital and Dr. Naeye's November 1999 report. Dr. Perper reiterated his opinion that Miner suffered from clinical and legal pneumoconiosis. He reaffirmed his prior opinion that Miner's death was hastened or contributed to by Miner's CWP based on Miner's long occupational exposure to coal dust, Miner's

respiratory symptomatology, the objective evidence of primarily obstructive lung disease and hypoxemia, and the resulting cor pulmonale with evidence of severe pulmonary hypertension, the significant CWP demonstrated at autopsy, and the associated severe centrilobular emphysema. Dr. Perper then identified direct and indirect evidence that served as competent evidence that Miner's CWP was a significant contributory cause of death: (1) direct replacement of normal lung tissue with pneumoconiotic lesions and associated centrilobular chronic emphysema and resulting hypoxemia; and (2) direct pulmonary insufficiency to a normally breathing lung by non-breathing pneumoconiotic tissues and associated centrilobular emphysema and resulting hypoxemia, and through hypoxemia precipitating/aggravating a cardiac arrhythmia in an individual withy heart disease. He also identified a scientific article finding that patients with COPD, especially during acute exacerbations, show a greater incidence of cardiac arrhythmia than healthy subjects of the same age.

Dr. Perper set forth clinical and pathological observations and findings based on Miner's medical records, the autopsy report, the autopsy slide, and his review of Dr. Naeye's opinion. He considered an accurate account of Miner's prior medical history and Miner's smoking and coal dust exposure history. I determined that Dr. Perper's finding of legal and clinical pneumoconiosis was better supported by the autopsy evidence than the opinions of Drs. Naeye and Repsher, who found that Miner only suffered from mild histological clinical pneumoconiosis. Thus, Dr. Perper considered the full extent of Miner's clinical and legal pneumoconiosis when he rendered his opinion on the cause of Miner's death. While Dr. Perper's August 1999 opinion identifying "the most likely mechanism of death by which pneumoconiosis contributed to death . . . " was curiously worded, Dr. Perper reaffirmed his conviction to that opinion and supported it with adequate clinical and pathological findings. He credibly defended his opinion from criticism levied by Drs. Naeye and Repsher by identifying his pathological findings and Dr. Hardy's pathological findings that supported his opinion that Miner's centrilobular emphysema was a significantly contributed to by Miner's coal dust exposure. Dr. Perper's opinion is reasoned and documented. I find that Dr. Perper's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical, anatomical, and forensic pathologist and by the fact that his opinion on the cause of Miner's death considered the full extent of Miner's pneumoconiosis.

Dr. Naeye rendered an initial opinion discussing the cause of Miner's death in November 1999. He rendered postmortem findings that Miner suffered from mild simple CWP, severe centrilobular emphysema, and chronic bronchitis. Dr. Naeye noted that Miner's death certificate attributed Miner's death to a combination of cardiac failure, chronic cor pulmonale, and COPD. From his review of the autopsy slides of Miner's heart and lungs, he opined that the enlargement of Miner's right ventricular wall was not the result of CWP causing cor pulmonale because Miner's CWP was far too mild to cause this effect. Instead, he attributed the cor pulmonale to Miner's centrilobular emphysema, chronic hypertension, and resulting left heart failure, which increased Miner's pulmonary venous blood pressure. Dr. Naeye concluded that Miner's CWP did not hasten his death due to its mild nature. Dr. Naeye devoted substantial attention to his opinion that Miner's severe centrilobular emphysema was not contributed to by Miner's coal dust exposure. He noted that he has reviewed the lungs of many miners of the one coal seam in United States' soft coal regions whose exposures to dust had made a major contribution to their emphysema and disability, but he found that Miner was not one of those miners based on his

review of the autopsy slides. He then reiterated his opinion that Miner's CWP was far too mild to have hastened or had any other role in Miner's death. Dr. Naeye submitted a supplemental report in June 2003 to respond to three specific criticisms of his report levied by Dr. Perper. He did not address the etiology of Miner's death in his supplemental report. Instead, he disagreed with his characterization of Dr. Perper's report that Dr. Perper diagnosed complicated pneumoconiosis and he disagreed that any very tiny birefringent crystals of toxic silica were present in Miner's lungs at autopsy. In his two reports, Dr. Naeye devoted considerable effort to supporting his diagnosis of mild clinical pneumoconiosis and refuting Dr. Perper's diagnosis of legal pneumoconiosis in the form of centrilobular emphysema arising out of coal mine employment. His only opinion on Miner's cause of death was limited to finding that Miner's clinical pneumoconiosis was far too mild to have hastened or caused Miner's death. Dr. Naeye did not provide an opinion on the actual cause of Miner' death, he only noted what Dr. Potter identified on the death certificate. He determined that Miner's centrilobular emphysema was a contributing cause leading to the development of Miner's cor pulmonale, which Dr. Potter had identified as a factor that combined with other factors to serve as the immediate cause of Miner's death. His opinion that Miner's CWP did not cause Miner's cor pulmonale was based on his limited opinion that Miner only suffered from mild clinical pneumoconiosis. An underlying predicate of Dr. Naeye's opinion that Miner's simple clinical pneumoconiosis did not cause Miner's cor pulmonale was that Miner's coal dust exposure was not a contributing cause of Miner's severe centrilobular emphysema. That underlying factual predicate is contrary the undersigned's determination that the preponderance of the autopsy evidence establishes that Miner's coal dust exposure was a significant contributing cause of Miner's centrilobular emphysema. Dr. Naeye's opinion excluding Miner's CWP as a causal factor in Miner's death partially based on a factual predicate contrary to the undersigned's determination regarding the extent of Miner's CWP. Dr. Naeye did not provide an opinion on the role of Miner's legal pneumoconiosis in relation to Miner's death. Therefore, I accord a limited degree of probative weight to the opinion of Dr. Naeye regarding the cause of Miner's death because he did not address the role of Miner's legal pneumoconiosis, if any, in causing Miner's death. In so doing, I recognize Dr. Naeye's credentials as a board-certified clinical and anatomical pathologist who has vast experience evaluating the lungs of coal miners.

Dr. King issued an opinion discussing the cause of Miner's death in June 2003. He reviewed his office notes from the time he served as Miner's treating physician (1991 to 1997), Dr. Hardy's autopsy report, and he spoke with Dr. Hardy. Dr. King opined that Miner's main disability was emphysema and pulmonary hypertension, mostly caused by cigarette use but with coal dust exposure also serving as a significant contributing factor to Miner's emphysema. He stated that Miner's CWP, which was proven at autopsy, contributed to Miner's death. Dr. King stated that Miner's nodular formation of CWP, which Dr. Hardy considered to be moderate, altered the normal lung architecture of Miner's lungs, thereby contributing to the development of Miner's hypoxia. Dr. King opined that Miner's hypoxia and altered lung structure led to pulmonary hypertension, which caused the dilation and hypertrophy of Miner's heart identified at autopsy. From that, Dr. King concluded that Miner's damaged heart was less able to handle an ischemic insult. Thus, he determined that the main cause of Miner's death was his emphysema and pulmonary hypertension, to which Miner's CWP was a contributory factor. Dr. King set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Miner's smoking and coal mine employment

histories. He addressed the primary disease processes present prior to Miner's death. His opinion is reasoned and documented. I previously accorded Dr. King's opinion enhanced probative weight, since it was reasoned and documented, on the issue of the etiology of Miner's pulmonary disability. However, I decline to grant any degree of enhanced probative weight to Dr. King's opinion based on his status as Miner's former treating physician. Dr. King last examined Miner in October 1997. Dr. Poitras, who followed Miner in the weeks just prior to Miner's death, commented that Miner underwent an inexorable decline in the past few years. Thus, even though Dr. King had examined Miner seven months prior to his death, there is no evidence in the record that Dr. King's treatment of Miner afforded him with superior and relevant information regarding the cause of Miner's death. His opinion on Miner's death was based on his treatment records and his review of the autopsy report. His treatment of Miner provided him with unique insight on the etiology of Miner's disability during his lifetime, but all other physicians who rendered an opinion on the cause of Miner's death were privy to the results of Dr. Hardy's autopsy. Thus, I find that Dr. King's opinion is entitled to probative weight enhanced by his board-certification in internal medicine.

The final opinion on the cause of Miner's death was issued by Dr. Repsher in his June 2003 report and his testimony at the formal hearing in July 2003. He reviewed Miner's autopsy report, the death certificate, and the reports of Drs. Perper, Naeye, and Hill. Dr. Repsher determined that Miner suffered from mild histologic CWP that was probably of no significance. He also found that Miner had COPD with severe centrilobular emphysema and severe cor pulmonale, as the result of Miner's long and heavy cigarette smoking habit. With regard to Miner's heart, Dr. Repsher noted that Miner had hypertensive cardiovascular disease probably with diastolic congestive heart failure and coronary artery disease. He also noted that miner had peptic ulcer disease with a recurrent upper gastrointestinal hemorrhage. Dr. Repsher opined that Miner's death was not hastened by his mild histological simple CWP because his CWP was so mild that it was not radiographically visible, Miner's death certificate did not even mention CWP, and Drs. Perper and Naeye agreed that Miner only had slight or moderate histologic CWP. These findings led Dr. Repsher to conclude that the amount of Miner's lung tissue involved with CWP would have been so small that it would not be anticipated that it would have any effect on Miner's lung function. Since there was no effect on lung function, Dr. Repsher anticipated to a reasonable degree of medical certainty that Miner's mild histologic CWP would not have caused, contributed to, or hastened Miner's death. Instead, Dr. Repsher opined that Miner's death was clearly due to many of the overwhelming deleterious effects of cigarette smoking and in no discernable way to Miner's CWP. Dr. Repsher reiterated his findings through his testimony at the hearing. He argued that Dr. Preger's x-ray interpretation was not consistent with pneumoconiosis arising out of coal mine employment, since he found lineal opacities instead of rounded opacities. Dr. Repsher testified that Miner died in his sleep, likely due to a heart attack or malignant arrhythmia (a fatal irregular beating of the heart). He added that Miner's pulmonary disability had no relation to Miner's heart attack or heart disease that caused his death. Dr. Repsher allowed that it was possible, although not probable, that Miner's arrhythmia was triggered by an event of hypoxia caused by severe COPD. He also testified that the most common cause of cor pulmonale is left ventricular heart disease, which is associated with coronary artery disease. His testimony concluded with his finding that Miner's death was not hastened by his simple CWP to any extent because the amount of CWP was so small that its effect on lung function would be too small to measure. Dr. Repsher set forth clinical

observations and findings. He considered an accurate account of Miner's smoking and coal mine employment history.

There are several problems with Dr. Repsher June 2003 report and his testimony based thereon. First, the three reasons Dr. Repsher identified in support of his conclusion that Miner's CWP did not cause, contribute to, or hasten Miner's death are not supported by the record. Dr. Repsher stated that Miner's pneumoconiosis was so mild that it was not even radiographically visible, but he acknowledged that he did not review Dr. Preger's positive interpretation of the 1996 x-ray film when he made that finding in his June 2003 report. Dr. Repsher acknowledged that he did not know that Dr. Potter did not have the results of the autopsy available to him when he signed the death certificate. Since the autopsy evidence revealed evidence of clinical and legal pneumoconiosis, the probative weight of the death certificate is limited. Dr. Repsher's third reason, his summary that Drs. Perper and Naeye agreed that Miner only had slight or moderate histologic CWP is misleading. While Drs. Perper and Naeye did only find a mild-moderate degree of clinical pneumoconiosis, the preponderance of the evidence also established that Miner also had legal pneumoconiosis in the form of severe centrilobular emphysema that was significantly contributed to by Miner's coal dust exposure. The findings that Dr. Repsher relied upon in his June 2003 report to state that Miner's death was not caused, contributed to, or hastened by are not adequate to support his conclusion. During his testimony, he attempted to rehabilitate that the adequacy of his findings, in part by stating that Dr. Preger's interpretation was not consistent with pneumoconiosis arising out of coal mine employment. While Dr. Repsher's analysis regarding Dr. Preger's interpretation, the autopsy findings revealed the presence of rounded opacities consistent with coal workers' pneumoconiosis and changes consistent with cigarette smoking, but there was no evidence of asbestosis. Dr. Repsher is not a pathologist and he did not review the autopsy slides. When rendering his June 2003 report, he did not even review Miner's actual medical records. He testified at the hearing that he relied upon Dr. Perper's summary of the medical records. While there are several minor problems with Dr. Repsher's opinion, his opinion suffers from one major flaw. Dr. Repsher's opinion on the etiology of Miner's death failed to consider the full extent of Miner's pneumoconiosis. His opinion was predicated solely on the lack of effect Miner's mild clinical pneumoconiosis had on Miner's pulmonary functioning. However, the undersigned determined that the autopsy evidence demonstrated that Miner suffered from legal pneumoconiosis as well. Dr. Repsher's opinion that Miner's CWP was too mild to play any role in causing, contributing to, or hastening Miner's death is undercut be his failure to consider the full extent of Miner's CWP. Therefore, I accord a limited degree of probative weight to the opinion of Dr. Repsher. In so doing, I recognize his credentials as a board-certified pulmonologist.

The death certificate attributed Miner's death to cardiac arrest, with underlying causes of cardiomegaly, chronic cor pulmonale, and COPD. Dr. Poitras, who treated Miner just prior to his death, noted that Miner had undergone an inexorable downhill decline over the past few years. When he discharged Miner to the Parkdale Care Center, Dr. Poitras diagnosed gastrointestinal bleed, congestive heart failure, COPD, and dementia. Dr. Hardy, the autopsy prosector who had access to some of Miner's hospital records, could not definitively determine the cause of death, but he suggested that dysrhythmia secondary to ischemic heart disease was a possible cause of death due to the degree of Miner's ischemic heart disease. However, he could not exclude the possibility of an intracranial process or a gastric/colonic bleed. Drs. Perper and

King, the only two physicians to consider the full extent of Miner's pneumoconiosis, determined that Miner's CWP was a substantial contributing cause of Miner's death. Dr. Perper opined that the most likely mechanism by which Miner's pneumoconiosis contributed to Miner's death was by causing hypoxemia that triggered or aggravated an arrhythmia caused by Miner's ischemic heart disease. Dr. Perper, in the final section of his initial report, concluded that the primary cause of Miner's death was the result of a combination of severe heart disease and cardiomegaly with severe COPD and simple CWP. In his supplemental report, Dr. Perper identified the evidence that he relied upon to conclude that Miner's CWP contributed to or hastened his death. Dr. Perper found that Miner's pneumoconiotic lesions and associated chronic centrilobular emphysema replaced normal lung tissue, causing hypoxemia. He then found that the hypoxemia precipitated or aggravated a cardiac arrhythmia in an individual with heart disease. Dr. Perper then identified a scientific article that found a higher incidence of cardiac arrhythmia patients with COPD than healthy subjects. Dr. King found that Miner's nodular CWP altered Miner's lung architecture, which contributed to the development of Miner's hypoxia. In turn, he found that Miner's hypoxia caused pulmonary hypertension, which caused the dilation and hypertrophy of Miner's heart. Dr. King found that Miner's damaged heart was less able to handle an ischemic insult. Dr. King found that Miner's coal dust exposure contributed to the development of Miner's emphysema. He then determined that Miner's emphysema and pulmonary hypertension were the two primary causes of Miner's death. Thus, he found that Miner's CWP was a significant contributing factor to Miner's death. Dr. Repsher testified that the mechanism of death described by Dr. Perper was possible, but not probable. Dr. Repsher opined that Miner likely died in his sleep due to a heart attack or malignant arrhythmia. Dr. Naeye did not offer an opinion on the exact cause of Miner's death. His opinion was focused on ruling out Miner's clinical pneumoconiosis as a cause of death. Dr. Naeye, after noting that the death certificate listed cor pulmonale as an underlying cause of death, found that Miner's severe centrilobular emphysema, chronic hypertension and resulting left heart failure caused Miner's cor pulmonale. The undersigned has determined that Miner's coal dust exposure contributed to the development of Miner's severe centrilobular emphysema. Even though Dr. Naeye opined that Miner's coal dust exposure did not cause his severe centrilobular emphysema, his opinion establishes a connection between Miner's legal pneumoconiosis and the development of his cor pulmonale.

I find that the preponderance of the reasoned medical opinions establishes that Miner's clinical and legal pneumoconiosis was a substantially contributing cause of Miner's death. In reliance on the death certificate, the opinion of Dr. Poitras, and the conclusions of Drs. Hardy, Naeye, Perper, King, and Repsher, I find that Miner's death was due to cardiac arrest brought about by a cardiac arrhythmia. Drs. Perper and King determined that Miner's clinical and legal pneumoconiosis, in conjunction with Miner's smoking history, caused the development of hypoxemia. Miner's hypoxemia led to the development of pulmonary hypertension. Dr. Hardy identified severe pulmonary hypertension at Miner's autopsy, and Drs. Perper and Naeye agreed with that finding. Drs. Perper and King, with support from Dr. Naeye, also found that Miner's severe centrilobular emphysema contributed to the development of Miner's cor pulmonale with right-sided congestive heart failure. Dr. Repsher stated that the most common cause of right-sided congestive heart failure is left-sided congestive heart failure caused by coronary artery disease, a condition that had been diagnosed in Miner. Dr. Naeye also found that Miner's left-sided heart failure also contributed to Miner's cor pulmonale. It is reasonable to conclude that Miner's congestive heart failure was brought about by his coronary artery disease (chronic

hypertension) and by his severe centrilobular emphysema. Dr. King opined that Miner's weakened heart was less able to withstand an ischemic insult. The preponderance of the evidence establishes that Miner's clinical and legal pneumoconiosis substantially contributed to Miner's due to cardiac arrest by causing hypoxemia, cor pulmonale, and pulmonary hypertension that led to the inducement of a cardiac arrhythmia that Miner's heart, weakened by conditions to which Miner's clinical and legal pneumoconiosis contributed to, could not withstand. Therefore, I find that Claimant has established that Miner's pneumoconiosis arising out of coal mine employment was a substantially contributing cause of Miner's death under § 718.205(c)(2). Therefore,

Entitlement

Claimant, Mary Kourianakis, has proven by a preponderance of the evidence, that Miner's death was due to pneumoconiosis arising out of coal mine employment. Emmanuel Kourianakis died in May 1998. Therefore, Mrs. Kourianakis is entitled to benefits under the Act beginning with May 1998. § 725.503(c).

Attorney's Fees

No award of attorney's fees for services to Mrs. Kourianakis is made herein, since no application has been received from counsel. A period of 30 days is hereby allowed for Mrs. Kourianakis' counsel to submit an application, with a service sheet showing that service has been made upon all parties, including Claimant. The Parties have 10 days following receipt of any such application within which to file their objections. The Act prohibits the charging of any fee in the absence of such approval. *See,* §§ 725.365 and 725.366.

ORDER

IT IS ORDERED that the claim of Mary Kourianakis for benefits on behalf of Emmanuel Kourianakis and for survivor benefits under the Act is hereby GRANTED.

A

THOMAS F. PHALEN, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.**